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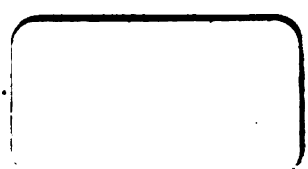

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TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Sixty-Seventh Annual
Session, held at Detroit,
Michigan, June 13 to 16, 1916

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LIST OF OFFICERS

This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the Secretary of the American Medical Association.

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1866 Chairman, C. A. Walker, Boston.

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Secretary, C. A. Lee, Peekskill, N. Y.

1869 Records wanting.

1870 Chairman, J. P. Gray, Utica, N. Y.
Secretary, A. Hartmann, Baltimore.

1871 Records wanting.

1872 Chairman, Isaac Ray, Philadelphia.
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1877 Chairman, W. Kempster, Oshkosh, Wis.
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Secretary, T. F. Wood, Wilmington, N. C.

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1884 Chairman, E. W. Schaufler, Kansas City, Mo.
Secretary, J. N. McCormack, Bowling Green, Ky.

1885 Chairman, J. H. Rauch, Chicago.
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Secretary, F. S. Bascom, Salt Lake City.

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Secretary, F. S. Bascom, Salt Lake City.

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1896 Chairman, Elmer Lee, Chicago.
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1898 Chairman, A. R. Reynolds, Chicago.
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1899 Chairman, W. C. Woodward, Washington, D. C.
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1888 Chairman, J. G. Kiernan, Chicago.
Secretary, T. B. Evans, Baltimore.

1889 Chairman, T. B. Evans, Baltimore.
Secretary, T. D. Crothers, Hartford, Conn.

1890 Chairman, T. D. Crothers, Hartford, Conn.
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1893 Chairman, J. G. Kiernan, Chicago.
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1894 Chairman, D. R. Brower, Chicago.
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1895 Chairman, T. D. Crothers, Hartford, Conn.
Secretary, W. J. Herdman, Ann Arbor, Mich.

1896 Chairman, W. J. Herdman, Ann Arbor, Mich.
Secretary, C. H. Hughes, St. Louis.

1897 Chairman, C. H. Hughes, St. Louis.
Secretary, H. T. Patrick, Chicago.

1898 Chairman, F. Peterson, New York.
Secretary, H. T. Patrick, Chicago.

1899 Chairman, H. T. Patrick, Chicago.
Secretary, F. S. Pearce, Philadelphia.

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1900 Chairman, H. A. Tomlinson, St. Peter, Minn.
Secretary, F. Savary Pearce, Philadelphia.

1901 Chairman, R. Dewey, Milwaukee, Wis.
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Delegate, F. W. Langdon, Cincinnati.

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Secretary, David I. Wolfstein, Cincinnati.

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Delegate, Morton Prince, Boston.

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Secretary, William Alexander Jones, Minneapolis.
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1909 Chairman, Theodore Diller, Pittsburgh.
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Delegate, M. Allen Starr, New York.

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Vice Chairman, Herman H. Hoppe, Cincinnati.
Secretary, E. E. Southard, Cambridge, Mass.

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Vice Chairman, George H. Moody, San Antonio, Tex.
Secretary, E. E. Southard, Cambridge, Mass.
Delegate, W. A. Jones, Minneapolis.

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Vice Chairman, W. W. Graves, St. Louis.
Secretary, G. A. Moleen, Denver.
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1913 Chairman, W. W. Graves, St. Louis.
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Vice Chairman, H. G. Brainerd, Los Angeles.
Secretary, George A. Moleen, Denver.
Delegate, W. W. Graves, St. Louis.

1915 Chairman, George A. Moleen, Denver.
Vice Chairman, M. A. Bliss, St. Louis.
Secretary, A. S. Hamilton, Minneapolis.
Delegate, Milton B. Lennon, San Francisco.

1916 Chairman, Bernard Sachs, New York.
Vice Chairman, Charles W. Hitchcock, Detroit.
Secretary, A. S. Hamilton, Minneapolis.
Delegate, George A. Moleen, Denver.

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PROCEEDINGS OF THE SECTION

TUESDAY, JUNE 13—AFTERNOON

The section was called to order by the chairman, Dr. George A. Moleen, Denver.

Dr. George A. Moleen, Denver, read the chairman's address, entitled "The Nervous System as Influenced by High Altitudes."

Dr. Peter Bassoe, Chicago, read a paper on "Tumors of the Third and Fourth Ventricles," with lantern demonstration. Discussed by Drs. Theodore H. Weisenburg, Philadelphia; Tom A. Williams, Washington, D. C.; Harold N. Moyer, Chicago; Lawson G. Lowrey, Hathorne, Mass.; A. L. Skoog, Kansas City, Mo., and Peter Bassoe, Chicago.

The chairman appointed the following members to act as a nominating committee: Drs. Theodore H. Weisenburg, Philadelphia, chairman; C. Eugene Riggs, St. Paul, and Walter F. Schaller, San Francisco.

Dr. R. Foster Kennedy, New York, read a paper on "A Further Note on the Diagnostic Value of Retrobulbar Neuritis in Expanding Lesions of the Frontal Lobes." Discussed by Drs. Francis R. Fry, St. Louis, and R. Foster Kennedy, New York.

The chairman referred to the recommendation received from the secretary of the Council on Scientific Assembly that the Section on Nervous and Mental Diseases be made a part of a general section on medicine with a view to condensation of the number of sections, stating that it would be well for those interested in the work of the section to give the matter thought, and in case any action were to be taken in the way of recommendations to the House of Delegates, he would be glad to entertain a motion to that effect.

Dr. Theodore H. Weisenburg, Philadelphia, read a paper on "A Clinical Study of Unusual Symptoms in Hemiplegia and Diplegia." Discussed by Francis X. Dercum, Philadelphia; Walter Timme, New York; George A. Moleen, Denver; J. Ramsay Hunt, New York, and Theodore H. Weisenburg, Philadelphia.

Dr. Charles K. Mills, Philadelphia, read a paper on "Some Clinical Studies of the Problems of Cerebral Tone." Discussed by Drs. William J. Hickson, Chicago; Julius Grinker, Chicago; R. Foster Kennedy, New York; J. Ramsay Hunt, New York, and Charles K. Mills, Philadelphia.

On motion, carried, the subject of the condensation of this section with a general section on medicine was made a

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special order of business to be considered at the termination of the scientific program Wednesday morning.

Dr. I. Leon Meyers, Chicago, read a paper on "Cerebellar Localization: An Experimental Study by a New Method." Discussed by Drs. Julius Grinker, Chicago; Charles K. Mills, Philadelphia; Tom A. Williams, Washington, D. C., and I. Leon Meyers, Chicago.

Wednesday, June 14—Morning

The chairman announced that, in order to expedite matters, it had been suggested that a committee be appointed for the purpose of formulating and presenting to this body for its consideration a resolution with reference to the objections entertained by the members to condensation of this section with a general section on medicine.

Dr. Charles K. Mills, Philadelphia, moved that a committee of three be appointed to prepare such resolution, same to be presented at the end of this session. Motion carried.

The chairman appointed as such committee Drs. Francis X. Dercum, Philadelphia; C. Eugene Riggs, St. Paul, and J. Ramsay Hunt, New York.

Dr. Charles K. Mills further moved that the delegate of this section, Dr. J. H. McBride, Pasadena, Calif., be added to the committee named, making four on the committee. Motion carried. Whereupon the chairman added Dr. J. H. McBride to the committee as named by him.

Dr. Francis X. Dercum, Philadelphia, read a paper on "Epilepsy, with Special Reference to Treatment." Discussed by Drs. George A. Moleen, Denver; David T. Weeks, Skillman, N. J.; A. L. Skoog, Kansas City, Mo.; Francis R. Fry, St. Louis; Charles R. Ball, St. Paul; William Ravine, Cincinnati; Julius Grinker, Chicago; R. A. Greene, Palmer, Mass.; James H. McBride, Pasadena, Calif.; Joseph M. Aikin, Omaha; William J. Hickson, Chicago; Francis A. Ely, Des Moines, Ia.; I. Leon Meyers, Chicago; D. S. Booth, St. Louis; Robert McGregor, Saginaw, Mich.; J. Matthew Pulliam, Fort Wayne, Ind.; R. Foster Kennedy, New York; Walter Timme, New York; L. Kauffman, Buffalo, and Francis X. Dercum, Philadelphia.

The chairman announced that the National Association for the Study of Epilepsy had sent a cordial invitation to members of the section to attend a meeting of the association at the Hotel Tuller, Friday, June 16, at 10 a. m. and 2 p. m.

Dr. J. Ramsay Hunt, New York, read a paper on "The Progressive Torsion Spasm (Dystonia Musculorum Deformans)." Discussed by Drs. Hugh T. Patrick, Chicago; E. W. Haass, Detroit (with exhibition of patient); A. L. Skoog, Kansas City, and J. Ramsay Hunt, New York.

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Dr. Hugh T. Patrick, Chicago, read a paper on "The Factor of Fear in Nervous Cases." Discussed by Drs. Frank P. Norbury, Springfield, Ill.; Harold N. Moyer, Chicago; Robert McGregor, Saginaw, Mich.; Walter B. Cannon, Boston; Tom A. Williams, Washington, D. C.; Walter B. Swift, Boston; R. Foster Kennedy, New York; Francis R. Fry, St. Louis; Charles R. Ball, St. Paul; U. G. Darling, Chicago; D. S. Booth St. Louis, and Hugh T. Patrick, Chicago.

Dr. J. Ramsay Hunt, New York, member of the committee appointed to prepare a resolution and report to the section in the matter of the proposed amalgamation of the section with the Section on Medicine, presented a report which was referred to the House of Delegates.

Wednesday, June 14—Afternoon

The chairman announced that the report of committee which was presented as the last order of business at the morning session would be placed in the hands of the delegate from this section for presentation to the House of Delegates [not presented to the House of Delegates].

Dr. A. M. Barrett, Ann Arbor, Mich., read a paper on "Syphilitic Psychoses Associated with Manic Depressive Symptoms and Course." Discussed by Drs. Frank P. Norbury, Springfield, Ill.; Hugh T. Patrick, Chicago; W. F. Lorenz, Mendota, Wis.; Milton Board, Louisville, Ky.; Julius Grinker, Chicago, and C. Eugene Riggs, St. Paul.

Drs. James B. Ayer and Henry R. Viets, Boston, presented a paper on "Spinal Fluid Findings Characteristic of Cord Compression," read by Dr. Ayer. Discussed by Drs. Peter Bassoe, Chicago; Francis A. Ely, Des Moines; A. L. Skoog, Kansas City; Ernest Sachs, St. Louis; Charles R. Ball, St. Paul; W. F. Lorenz, Mendota, Wis.; Charles A. Elsberg, New York, and James B. Ayer, Boston.

Dr. Morris J. Karpas, New York, read a paper on "Constitutional Inferiority in the Light of Modern Conceptions." Discussed by Drs. Frank P. Norbury, Springfield, Ill., and Morris J. Karpas, New York.

Dr. Lewis J. Pollock, Chicago, read a paper on "Experimental Studies of Injection of the Gasserian Ganglion Controlled by Fluoroscopy." Discussed by Drs. Hugh T. Patrick, Chicago; Julius Grinker, Chicago; M. A. Bliss, St. Louis, Mo.; Charles R. Ball, St. Paul; Ernest Sachs, St. Louis; George M. Dorrance, Philadelphia, and Lewis J. Pollock, Chicago.

Dr. George M. Dorrance, Philadelphia, read a paper on "A New Method of Injecting the Facial Nerve for Relief of Facial Spasm." Discussed by Drs. Hugh T. Patrick, Chicago; A. L. Skoog, Kansas City; Julius Grinker, Chicago, and George M. Dorrance, Philadelphia.

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Dr. William A. Jones, Minneapolis, read a paper on "Myasthenia Gravis." Discussed by Drs. Walter B. Swift, Boston; R. Foster Kennedy, New York; C. Eugene Riggs, St. Paul; George A. Moleen, Denver, and William A. Jones, Minneapolis.

Drs. Pearce Bailey and Walter Timme, New York, presented a paper on "A Contribution to the Pathogenesis of Progressive Muscular Dystrophy, with the Consideration of Evidence Connecting this Disease with Disturbances in the Endocrine Glands," read by Dr. Timme. Discussed by Drs. A. L. Skoog, Kansas City; M. A. Bliss, St. Louis; Lewis J. Pollock, Chicago; George A. Moleen, Denver; Albert E. Sterne, Indianapolis, and Walter Timme, New York.

Thursday, June 15—Morning

The chairman announced the appointment of Dr. William A. Jones, Minneapolis, on the Executive Committee, to take the place of Dr. Francis X. Dercum, who had found it necessary to retire; and that therefore, for the remainder of the session, papers would be approved by Dr. Jones as member of the Executive Committee.

The following officers were elected for the ensuing year: chairman, Dr. Bernard Sachs, New York; vice chairman, Dr. Charles W. Hitchcock, Detroit; secretary, Dr. A. S. Hamilton, Minneapolis; delegate, Dr. George A. Moleen, Denver.

Dr. Harry H. Drysdale, Cleveland, read a paper on "Conjugal Paresis: Report of a Case." Discussed by Drs. Julius Grinker, Chicago; J. Matthew Pulliam, Fort Wayne, Ind.; Lawson G. Lowrey, Hathorne, Mass.; C. C. Burlingame, South Manchester, Conn.; William Ravine, Cincinnati; W. F. Lorenz, Mendota, Wis.; Charles R. Ball, St. Paul; G. H. Moody, San Antonio, Texas; S. G. Burnett, Kansas City, Mo.; Robert H. Haskell, Ionia, Mich.; George A. Moleen, Denver, and Harry H. Drysdale, Cleveland.

Dr. Henry F. Stoll, Hartford, Conn., read a paper on "Syphilis as a Cause of Chronic Invalidism." Discussed by Drs. W. W. Graves, St. Louis; A. L. Skoog, Kansas City; William Ravine, Cincinnati, and Henry F. Stoll, Hartford, Conn.

Dr. Walter F. Schaller, San Francisco, read a paper on "The Early Diagnosis of Tabes Dorsalis."

Drs. C. Eugene Riggs and Ernest M. Hammes, St. Paul, presented a paper on "Shall We Treat the Paretic?" read by Dr. Riggs.

Dr. Charles R. Ball, St. Paul, read a paper on "A Consideration of the Serobiologic Reactions after Five Years of Observation."

Dr. L. B. Pilsbury, Lincoln, Neb., read a paper on "Mercurial Medication with Spinal Drainage."

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These four papers were discussed by Drs. J. Henry Barbat, San Francisco; Tom A. Williams, Washington; Albert E. Sterne, Indianapolis; Julius Grinker, Chicago; Capt. M. A. Reasoner, Washington, D. C.; G. H. Moody, San Antonio, Texas; Henry F. Stoll, Hartford, Conn.; W. F. Lorenz, Mendota, Wis.; James B. Ayer, Boston; Robert H. Haskell, Ionia, Mich.; M. S. Woodbury, Clifton Springs, N. Y.; George A. Moleen, Denver; L. C. Bacon, St. Paul; Walter F. Schaller, San Francisco; C. Eugene Riggs, St. Paul; Charles R. Ball, St. Paul, and L. B. Pilsbury, Lincoln, Neb.

Thursday, June 15—Afternoon

Dr. Walter B. Swift, Boston, read a paper on "A New Treatment of Paralysis Agitans." No discussion.

Dr. William Fuller, Grand Rapids, Mich., read a paper on "Necessity of Revising the Nomenclature of the Anatomy of the Brain." Discussed by Drs. I. Leon Meyers, Chicago; William A. Jones, Minneapolis; Walter B. Swift, Boston; D. S. Booth, St. Louis, and William Fuller, Grand Rapids.

Dr. Tom A. Williams, Washington, read a paper on "A Contribution to the Pathogenesis of the Migraines: Notes Concerning a More Efficacious Treatment." Discussed by Drs. J. Matthew Pulliam, Fort Wayne, Ind.; J. C. De Venney, Harrisburg, Pa., and Tom A. Williams, Washington, D. C.

The chairman announced the appointment of Dr. Ross Moore, Los Angeles, on the Executive Committee, to take the place of Dr. W. W. Graves, who had been obliged to go away before the meeting adjourned.

A vote of thanks was extended to the members of the profession of the city of Detroit for the entertainment furnished and for the comfortable surroundings in which the meeting of the section had been held.

A vote of thanks was extended to the retiring chairman of the section for his efficient services in connection with the meeting, and also a vote of thanks was extended to the secretary, whose efforts had made possible the very exceptional arrangement of the program and who had secured papers exhibiting such great variety in subject and serving to maintain so much interest in the discussions.

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THE NERVOUS SYSTEM AS INFLUENCED BY HIGH ALTITUDES *

GEORGE A. MOLEEN, M.D.
DENVER

The call to order of this section marks the sixteenth year of its identity under its present name.

In taking the chair, I must first avail myself of the opportunity of expressing my thanks in appreciation of the honor of having been selected for this office and to assure you that it gives me much pleasure to preside at this meeting. In directing the proceedings and discussions I shall be dependent on your consideration, your cooperation and your assistance.

Prior to 1900, this branch of medicine was classified with some other sections. Consistent with the advance made in the study of diseases of the nervous system, its position as a special section was established and maintained since that time. In view of the recommendation that it be made a part of the Section on Medicine, I feel that intimate association places me in a position to enter a competent protest at this time, and that in so doing I am voicing the concurrence of the members interested in the work of this section. The place which this section has made for itself was merited and justified sixteen years ago and is now, and I trust it will continue as a special scientific section for the advancement of its purposes, and especially by reason of the possibility of wider dissemination of neurologic achievements for the general as well as the special medical professions enjoyed by this section.

Custom has always made it fall within the province of the chairman to present at the opening of the session some general subject in which he was particularly

* Chairman's address.

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interested. The one selected, while especially important with reference to certain localities, is not, I trust, without a claim to general interest. There is no literature, so far as I am aware, bearing directly, and not a great deal bearing indirectly, on the question involved.

The frequency with which individuals exhibit nervous symptoms, or complain of "nervousness" while living at increased elevations is responsible for the popular or common belief that one may expect to become "nervous" because one lives at the higher altitudes. This view has obtained so long that it is expressed with as little consideration as its value, empirically, really merits; but in the belief that a general *a priori* review of the underlying factors involved might be advantageously brought to the attention of the section, it was chosen as being of sufficient interest to form a part of the annual communication of the chairman and at the same time relieve it of an aspect of formality or conventionalism.

Some one has commented on the humor of the tendency in Colorado to charge to the altitude all of the evil or disagreeable changes experienced, while the climate is invariably credited with the benefits derived. Such expressions as "high altitudes are too hard on the nervous system" and "one cannot live any length of time at such an elevation without a nervous breakdown," and again, "women cannot live in Leadville (10,200 feet) because their nervous system does not seem to stand the altitude—they become nervous wrecks" are merely the result of experiences and accepted by the public, and in no little part by the profession, without inquiry as to the reason for the manifestations commonly grouped under the term "nervousness" being brought about.

The first clue to a most important factor in determining these manifestations is to be found in the improvement frequently observed as a result of a more

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or less prolonged stay at a lower elevation or at the sea level—indeed, so often is this the case that the advice has become a routine. It would appear to be likewise significant that the plethoric type of individual rarely finds it necessary to leave the higher altitudes, while it is most often those evidencing a type of relative or absolute anemia who seem invariably to find relief at the lower elevations. A sufficient number of cases have been observed in which environmental influences could be eliminated as playing a negligible rôle.

It is not a definite syndrome with which we have to deal, but rather a state or condition of the nervous system probably best characterized as an irritability or hyperexcitability which may manifest itself in the motor, sensory or psychic spheres, or a combination of them, in an otherwise normal individual. Associated with this increased excitability is an increased rapidity of fatigue which finds expression in muscular weakness and diminished physical endurance, as well as failure in adaptability and power of concentration mentally. They complain of a mental unrest approaching anxiety, and find difficulty in carrying on the usual mental requirements of their occupations, owing to the failure to concentrate or sustain the attention. It is quite indicative when an individual experiences his best functional ability on awakening in the morning, which rapidly gives way to an unusual degree of fatigue, increasing as the day progresses. This condition may be the forerunner of a simple neurasthenia, or in the presence of a congenital predisposition the development of the more profound neuroses is facilitated. The psychic determinants of neurotic states find such a nervous system an easier prey to their influence.

If we turn our attention now to the purely physical facts in relation to altitude we find there is a progressive diminution in atmospheric pressure in accordance with the elevation. For example, at the sea level the

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barometer will register in the neighborhood of 760 mm. (30 inches) with a temperature of 50 degrees; at Denver (5,280 feet) under like conditions 630 mm. (24.74 inches) and at the summit of Pike's Peak (14,109 feet) from 452 to 462 mm. (17.8 to 18.2 inches). With the diminution of pressure, the atmospheric expansion is inversely proportionate and consequently the weight of oxygen in a given volume of air is correspondingly diminished.

Applying these facts physiologically, it is apparent that in order that a requisite amount of oxygen be transported, an increase in the volume of air inspired must take place; an increase in the lung area utilized; a greater number of corpuscles be exposed to aeration, and an increase in the amount of hemoglobin. The latter would seem to be required in even greater amount since, as Barcroft[1] has shown, the saturation power of hemoglobin varies with the oxygen pressure.

In 1911, the Anglo-American Pike's Peak Expedition, undertaken by members of Oxford and Yale universities, made extensive physiologic observations,[2] when it was found that "the percentage of hemoglobin in the blood increased for several weeks on the summit of Pike's Peak, and varied in various *acclimatized* persons from 115 to 154 per cent. on the scale of the Gowers-Haldane hemoglobinometer, corresponding to a percentage of oxygen capacity of from 21 to 28.5 c.c. of oxygen per 100 c.c. of blood. The number of red corpuscles increased parallel with the hemoglobin and there was no change in relation between the coloring power and oxygen capacity of the hemoglobin or in the dissociation curve of hemoglobin in the arterial blood. The increase in hemoglobin was apparently due to" concentration of the blood "for the first few days," but "afterwards entirely to a large increase in the total amount of hemoglobin." Barcroft,[3] in com-

1. Barcroft: Respiratory Function of the Blood, 1914, p. 15.
2. Phil. Tr. Roy. Soc. London, Series B, cciii, 185.
3. Barcroft: Respiratory Function of the Blood, Barcroft, 1914, p. 130.

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menting on his own observations and those just mentioned, concludes that "in each case there was a gradual rise in the total oxygen capacity, which reached its maximal value only after some time, about three weeks after the ascent. This perhaps is the essential point, for it proves quite clearly that the body reacts to the altitude either by producing increased quantities of hemoglobin or by retaining what would otherwise be broken." In this regard, however, the observations of Bürker[4] differ somewhat in that the individual variations were found to be greater. He found that smaller and lighter individuals reacted most strongly and more promptly; hemoglobin and erythrocyte count increased uniformly, so that the average hemoglobin content of the corpuscle underwent no essential change. In the larger and heavier individuals the hemoglobin content increased more than the numerical count so that the corpuscles were richer in hemoglobin in the higher elevations. The same author states that "The blood reaction as influenced by high altitudes is to be construed as an adaptation of the oxygen-carrying surface of the blood to the altered external conditions of the lower elevations, especially to the want of oxygen through the diminished atmospheric pressure."

The symptoms observed on ascending Pike's Peak, such as blueness of the lips and face, loss of appetite, nausea and vomiting, intestinal disturbances, headache, fainting in some persons, periodic breathing and great hyperpnea on exertion, are transitory manifestations and persist two or three days, when distinct signs of acclimatization become apparent. In this process of acclimatization, three factors became evident: (*a*) increased secretory activity of the lining cells of the lung alveoli; (*b*) lowering (in consequence of the diminished alkalinity of the blood) of the threshold of alveolar carbon dioxid pressure, and (*c*) increased percentage of hemoglobin in the blood. The stimulus

4. Bürker: Ztschr. f. Biol., 1913, lxi, 379.

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to these compensatory changes is deficient aeration of the blood passing through the lungs; and the compensation attained is, though very considerable, not complete.

Acclimatization in the normal individual presupposes, therefore, the ability to supply especially the required increase in the red blood cells and in the amount of hemoglobin. The degree of this ability then determines the completeness of acclimatization, and the oxygen want must obviously be in proportion to deficiency in the power of adaptability.

With this brief reference to the experimental work of Zuntz, Bürker, Barcroft, Haldane, Douglas, Henderson, Schneider and others, it is obvious that an increased activity of the blood-forming organs is the normal process of adaptation stimulated through oxygen want in the higher altitudes. Moreover, it becomes apparent that the clinical standard of corpuscular and hemoglobin determinations which might be regarded as within the normal at the sea level, when obtained at an elevation of 5,000 feet or more, may be regarded as a relative anemia which deserves consideration as a possible foundation for pathologic symptoms.

Now, as is well stated by Sewall[5]:

> The very essence of constitutional disease is the failure of physiological response on the part of living tissues to the external stimuli which are their normal excitants. Far from being strange, it is to be expected that in a certain proportion —a large proportion—of people the physiological response of the blood-forming tissues to the stimulus of altitude should be slow or imperfect. The clinical result is relative anemia.

Assuming, then, that anemia results fundamentally in the limiting of oxygen supply to the tissues, it seems proper to direct attention to the nervous tissue metabolism under like circumstances. The necessity for oxygen, and the fact that it is used up during the action of the brain,[6] can be very strikingly demonstrated by

5. Sewall: Colorado Med., 1915, xii, 41.
6. Halliburton: Biochemistry of Muscle and Nerve, 1904, p. 79.

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the experiment of Hill with methylene blue, and it can also be shown that this use is confined to the period of activity.

Thurnberg[7] showed that the consumption of oxygen and production of carbonic acid in nerve cells can be demonstrated directly by chemical analysis.

That individual ganglion cells present different resistances to anemia has been further confirmed by Landergren's[8] work on the phenomena of acute asphyxia; especially noteworthy is the finding of a brief rise of activity in the vasobulbar center.

Verworn's[9] work on the metabolism of nerve cells by means of strychninized frogs demonstrated that when the excitability was lost, the constituent in the blood responsible for restoration was not organic nutritive materials, but solely due to the oxygen. From his result he distinguishes two fundamentally different factors in the paralysis of the centers, namely, the fatigue due to the accumulation of the toxic products of metabolism and exhaustion due to the consumption of the supply of oxygen.

It can be proved in many ways that the passage of a nerve impulse through the nerve centers requires the expenditure of energy by these centers. It is significant that in all nervous systems, especially in the higher animals, that arrangements are made for their free supply of oxygen.[10] Very short deprivation of oxygen causes a complete block throughout the system, in many cases preceded by a short period of increased excitability or ease of transmission.

As early as 1879 Schmoulewitsch,[11] actuated by the experiment of Stevson, dating from the seventeenth century, found that deprivation of blood did not, as was previously observed, cause an immediate loss of

7. Thurnberg, quoted by Luciani: Human Physiology, 1915, iii, 231.
8. Landergren, referred to by Luciani: Human Physiology, 1915, iii, 267.
9. Verworn, referred to by Luciani: Human Physiology, 1915, iii, 271.
10. Starling: Human Physiology, 1915, p. 314.
11. Schmoulewitsch: Bull. gén. d. therap.; abstr., Periscope, Jour. Nerv. and Ment. Dis., 1879, vi, 154.

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irritability. On the contrary, this is for a while even increased and commences to disappear only after a certain degree of augmentation. The same phenomenon was observed after section of a nerve. The irritability of the corresponding muscle is increased for the first few moments. In his opinion, given at that time, this was to be attributed also to anemia, as the immediate result of nerve section. In this regard his conclusions are interesting:

> That the anemia is the cause of increased irritability of the muscles, I have proven by the following experiments:
>
> 1. After compression of the aorta or ligating the artery of a muscle, we cannot produce any augmentation of the irritability after section of a nerve. This demonstrates that this augmentation depends exclusively upon the circulation, for, as I say, the circulation once interrupted, the section of the nerve is without effect.
>
> 2. In curarizing an animal to the stage of complete paralysis, we always find an increase of muscular irritability following the section of the nerve. Here, evidently, only the vasomotor nerves can act, since, as has been demonstrated, they are not easily paralyzed by curare.
>
> Anemia, therefore, like certain affections of the nervous system that disorder the functions of the vasomotors, should increase the muscular irritability, a fact observed clinically, but not as yet sufficiently demonstrated theoretically.

It seems evident, therefore, that in the absence of oxygen supply the excitability of the protoplasm of nerve and other cells is lost—a fact which at first sight appears to be in conflict with clinical experience. It must be kept in mind, however, that we are concerned with a lessened oxygen supply, rather than with a complete withdrawal.

Is the increased irritability and consequent fatigue to be attributed to the insufficient oxygen supply primarily, or to the influence of the products of metabolism which have not been sufficiently oxidized to secure their elimination?

In order that expressions from more competent and authoritative sources might be presented, personal communications with the foremost physiologists were solicited and in general reveal an inclination to the

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former view, namely, that the irritability is to be charged to the diminution of oxygen supply through failure to effect the ordinary compensation. Professor Henderson considers that a "moderate deprivation of oxygen may readily cause an increase in irritability and a greater readiness to fatigue, while an extreme deprivation reduces irritability." He also calls attention to the fact that in the term "irritability" it is not an increase of functional power that is implied, but rather the reverse—an ease of excitability or irritability in what might be considered the popular rather than the scientific sense.

Dr. Haldane expresses himself as in accord with the writer, in that the hyperexcitability is to be attributed to oxygen want, and does not accept the view that oxygen want is sedative; and to emphasize his view, points out "that the effect of cutting off a man's victuals is not sedative—anyhow in the early stages." He further summarizes his views as: "1. Want of oxygen, within limits, is or may be excitant and not sedative. 2. The amount of oxygen in the blood is not an index of sufficiency at low pressures."

The unoxidized toxic or intermediary products of metabolism are preferably to be regarded as depressants, according to Dr. Howell, and in accordance with their concentration; although he cites the observation of Lee, that the first effect of the fatigue products of muscular contraction is an increase of irritability followed by depression when the concentration is presumed to have increased.

Without entering further into details with reference to the mechanism of the finer adjustments of the body to the oxygen supply, such as the carbonic acid or rather the hydrogen-ionic influence on the respiratory center, the relation of muscular exertion to its production, mode of formation, relations of alveolar and blood content and influences modifying these relations, etc., it seems established, that there is an irritability of the nervous system to be attributed to

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diminished oxygen supply by reason of the failure on the part of certain individuals to bring about an adaptation to the conditions incident to the diminished atmospheric pressure.

It must be admitted that there is good reason to suspect the possibility of some remote pathologic process as a fundamental factor inhibiting the power of adaptability in certain persons.

The estimation of hemoglobin by means of most of the ordinary methods in use depends on individual judgment of shades of color, and variations are often quite considerable. There is a strong tendency to read the percentage too high, especially when the examination is made in a subdued light. This was found to be particularly true when daylight was the source of illumination. In eighty-nine comparative observations made with day, and artificially lighted instruments, taking the judgments of three individuals, the estimations were invariably higher by the latter method, and varied from 6 to 15 per cent.

The corpuscular count was found to correspond so uniformly to the hemoglobin percentage that in the majority of cases it might be omitted.

As a result of several years' careful observations at an altitude of about 5,000 feet, it seems evident that the threshold of nervous stability varied between 82 and 88 per cent., corresponding to a corpuscular enumeration of from 4,100,000 to 4,400,000, and influenced in individual cases by demands made on the nervous structures as determining causes.

It was recognized quite early that the clinical ineffectiveness of the ordinary chalybeate therapeutic measures was not to be regarded as negativing the lowered blood values as a cause of the nervous irritability, since not infrequently subsequent estimations made in the absence of clinical improvement were found to show but little increase and were in some instances even lowered. It was necessary to change the form of iron administration, especially in the sta-

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tionary cases, until one was found to be effective in elevating the hemoglobin content. With this elevation the clinical improvement was uniformly constant, regardless of the means of accomplishment. In a few instances the removal to a lower altitude with the continuation of the iron administration proved effective when no impression was observed while at the higher elevation.

A marked increase in the elimination of the phosphates noted in a majority of the cases suggested the administration of the alkaline and earthy normal phosphates, and in some of the stationary cases seemed to augment the iron assimilation.

The question "Do so-called neurasthenic cases or those of 'nervousness' occur more frequently at high altitudes?" must be answered in the negative, but for several reasons. First, the general standard of living is better than in the more congested centers of population; second, the confined artificially lighted indoor workers are less common; third, there is a greater average of bright, cloudless days; lastly, the greater intensity or actinism of the light.

Finally, it is the class of patients who are able to live with more comfort at the lower altitudes and who manifest irritable neurotic disorders repeatedly on going to the higher elevations that prompted this investigation and as a result of which it may be concluded that:

1. The demand for oxygen-carrying elements of the blood increases directly with the altitude.

2. In normal individuals this requirement is met through an increase in the red blood corpuscles and hemoglobin in from three to five weeks—the normal acclimatization.

3. This power of adaptation is diminished or wanting in certain individuals.

4. Deficient acclimatization results in oxygen want or relative anemia.

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5. As a result of diminished or limited oxygen supply, the increased excitability or irritability of the nerve structures may be explained.

6. If by therapeutic or other means, the blood forming mechanism can be stimulated into activity, individuals should find no more difficulty in living tranquil lives in the high altitudes than at the sea levels.

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TUMORS OF THE THIRD AND FOURTH VENTRICLES

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I. TUMORS OF THE THIRD VENTRICLE

In 1910 T. H. Weisenburg,[1] on the basis of thirty reported cases with necropsy, succeeded in bringing out the salient features of tumors arising within the third ventricle and thus establishing a syndrome which necessarily is ill defined, as the symptoms are either due to pressure on neighboring structures or to secondary hydrocephalus. Among the former symptoms are paralysis of the associated ocular movements, exophthalmos, large pupils with impaired reaction, and ataxia of the cerebellar type. Of the few cases reported later, one by Lewis J. Pollock[2] may be mentioned, as the appearance of the tumor on cross section of the brain closely resembles that of the one to be related here.

CASE 1.—*Boy, 14 years old, with rather precocious genital and hairy development suggesting hyperpinealism. Limitation of ocular movements. Papillitis and blindness. Ataxia. Headache; drowsiness; mental dulness. Soft glioma filling third ventricle; secondary hydrocephalus.*

A boy 14 years old was admitted to the surgical service of Dr. Carl B. Davis in the Presbyterian Hospital, Aug. 24, 1915, having been referred to him by Dr. J. C. Bucher of Verona, Ill. The parents and two brothers and sisters are in good health. The patient had previously been healthy and had done well in school. He had been of a solitary and shut-in disposition and had used tobacco to excess for four or five years. Two months before admittance he had daily headache

1. Weisenburg, T. H.: Brain, 1910, xxxiii, 236.
2. Pollock, Lewis J.: Tumor of the Third Ventricle, THE JOURNAL A. M. A., June 5, 1915, p. 1903.

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for a week or ten days, then it subsided, and he had not complained of it for a month before coming to the hospital. Two weeks before admittance he suddenly became blind, and a few days later he had an epileptiform attack. From the time of the onset of headache he gradually grew dull mentally.

Examination by the writer on the day of admission: Well developed boy with healthy, ruddy complexion. The head is rather large, the circumference being 58.5 cm. He has a very distinct mustache for his age, also abundant pubic hair. He is dull, drowsy and apathetic and cannot give a reliable account of his previous history. The pupils are equal, wide and do not react to light or in accommodation. When at rest the eyeballs diverge a little. There is apparently no light perception. The movements of the eyeballs, especially the left one, are much limited. The left eye is only moved 15 degrees outward, the right one twice as much. Marked bilateral choked disk with hemorrhages. Corneal reflexes present. He lies on his back with head turned to the left and holds the neck stiff. Any movement of the head, especially to the right, is resisted and seems to cause headache. The gait is very unsteady and he has a tendency to fall backward. Ataxia of the extremities is demonstrated by the finger to nose and heel to knee tests. Examination of sensation is unsatisfactory, but apparently there is some diminution of pain sense on the left side of the face. The facial msucles appear somewhat weak on both sides, and the greatest weakness is found in the shutting of the left eye. The watch is heard in both ears. Knee jerks are absent; left ankle jerk absent, right present. Abdominal and cremasteric reflexes present. Wrist and elbow jerks present. No Babinski sign. The genitalia are very well developed.

Blood examination: Hemoglobin, 82 per cent.; red cells, 6,000,000; white cells, 15,900.

Urine: Normal except for excess of phosphates. The Wassermann test is negative with blood and spinal fluid, and the latter shows no increase in cells or globulin and gives a negative Lange colloidal gold test.

August 27, ophthalmoscopic examination by Dr. C. G. Darling shows a swelling of 8 diopters in the right eye and 4 or 5 in the left eye.

August 30: Getting worse mentally; seems disoriented; soils bed; has vomited his breakfast a few times; little headache, always tendency to fall backward.

A diagnosis of brain tumor was made, most likely located in the region of the corpora quadrigemina or pineal body, although the possibility of a cerebellar site could not be excluded. However, only a decompression operation was advised.

August 31: Right subtemporal decompression was made by Dr. Davis. The brain was found to be under unusually high pressure. The temperature, which previously had not

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exceeded 99 F., rose to 102.4 six hours after the operation, and reached 104.8 before death, which occurred thirty hours after the operation.

Necropsy.—The brain weighs 1,450 gm. The sulci are shallow and the gyri flattened. On the right side is a bulging area corresponding to the recent decompression operation. On the inferior surface there is a marked downward bulging in the region of the pineal body. The latter structure, however, is of normal appearance. After hardening in liquor formaldehydi the brain was cut by means of frontal sections. The bulging referred to is found to be caused by the great distention of the lateral ventricles. A soft, light gray tumor is found to fill the third ventricle (Fig. 1). Posteriorly it lies free in the ventricle, plugging the opening of the aqueduct, and appears to have a slight capsule. Its upper surface bulges into and completely closes the foramina of Monro (Fig. 2). In front of this level the tumor is seen to extend in a downward direction in front of the infundibulum, the anterior end being 9 mm. in front of the anterior border of the optic chiasma. The bulging at the base produced by this forward growth of the tumor begins at about the center of the base of the infundibulum and extends 2.3 cm. forward, or 1.1 cm. in front of the chiasma. The greatest anteroposterior measurement of the tumor in the third ventricle is 4.7 cm. Transversely it measures 1.9 cm. and vertically 2.1 cm. The degree of hydrocephalus may be judged from Figure 3, showing the anterior portion of the lateral ventricles.

Histologic Examination.—This tumor is very edematous, loose in texture, and in most places shows considerable evidence of tissue degeneration. Small hemorrhagic areas are seen. The cells are very variable in size and shape, and only in a few places is there any significant arrangement of them.

Many of the cells have the appearance of astrocytes, and very abundant fine fibers are seen in Van Giesen specimens. Their appearance suggests glia; but the Weigert glia stain failed, probably on account of the tissue degeneration. On the border of the tumor tissue remains of the ependymal layer of cells may be seen, in the form of palisade-like columns (Fig. 4).

II. TUMORS OF THE FOURTH VENTRICLE

Tumors of the fourth ventricle are much more common than those of the third ventricle. They also produce symptoms by inducing hydrocephalus and by exerting pressure on neighboring structures, in this case the cerebellum, pons and medulla. Like all other posterior fossa tumors, they may produce stiffness of the neck and occipital pain; and as increase in cells

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and globulin as well as in the pressure of the spinal fluid is common, a clinical resemblance to meningitis sometimes exists. This is well shown in our Case 2, while Case 3 presented the picture of cerebellar tumor, and in Cases 4 and 5 mental symptoms predominated. In another case reported by the writer[3] in which, in addition to the fourth ventricle tumor, another ependymal glioma in the frontal lobes was present, mental symptoms entirely dominated the clinical picture. Sudden death is very frequent in these cases.

CASE 2.—*Early symptoms suggestive of meningitis. Cloudy spinal fluid with increase in cells, both polymorphonuclear and mononuclear. Papillitis. Duration five and one-half months. Sudden death. Very large tumor in fourth ventricle, breaking through and almost surrounding pons.*

A boy 2½ years old, formerly well, was seen with Dr. H. F. Helmholz at the Evanston Hospital, Feb. 15, 1914. Ten weeks previously he had been taken ill with vomiting which occurred every day. There was light fever, the temperature occasionally reaching 101 F. The child seemed to suffer from headache. Strabismus and nystagmus had been noted, but there were no convulsions.

Blood examination, February 12: Hemoglobin, 75 per cent.; reds, 5,000,000; white cells, 15,000.

Examination.—Slight bilateral papillitis; no Kernig sign, no rigidity; knee and ankle jerks weak; abdominal reflexes weak, left weaker than right; doubtful left Babinski sign. The child was fairly bright and good natured. On making lumbar puncture the fluid was cloudy and escaped in a stream. There were 363 cells per cubic millimeter, about two thirds of which were polymorphonuclear. No bacteria were found in smears or cultures. As tuberculosis was suspected, a guinea-pig was inoculated but did not develop the disease.

For further information regarding this case I am entirely indebted to Dr. Helmholz and to Drs. W. O. Hoffman and A. B. Schwartz, in whose article on the permanganate reduction index of the cerebrospinal fluid[4] this case appears as Case 1. February 17, slight rigidity of the neck appeared with suggestion of the Kernig sign on the left side. There was a distinct Babinski sign on the left side; right plantar reflex normal. February 18, spinal fluid still opalescent but pressure not increased. The patient improved, and on February 24

3. Bassoe, Peter: Multiple Ependymal Glioma, Arch. Int. Med., September, 1908, p. 194.

4. Hoffman, W. O., and Schwartz, A. B.: The Permanganate Reduction Index of Cerebrospinal Fluid, Arch. Int. Med., February, 1916, p. 293.

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Fig. 1 (Case 1).—Tumor filling third ventricle.

Fig. 2 (Case 1).—Anterior portion of tumor.

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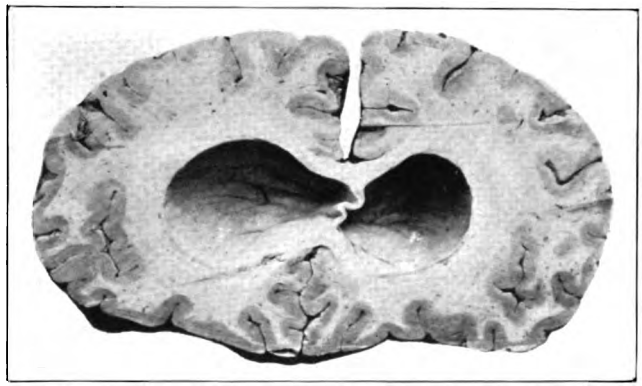

Fig. 3 (Case 1).—Anterior portion of distended lateral ventricles.

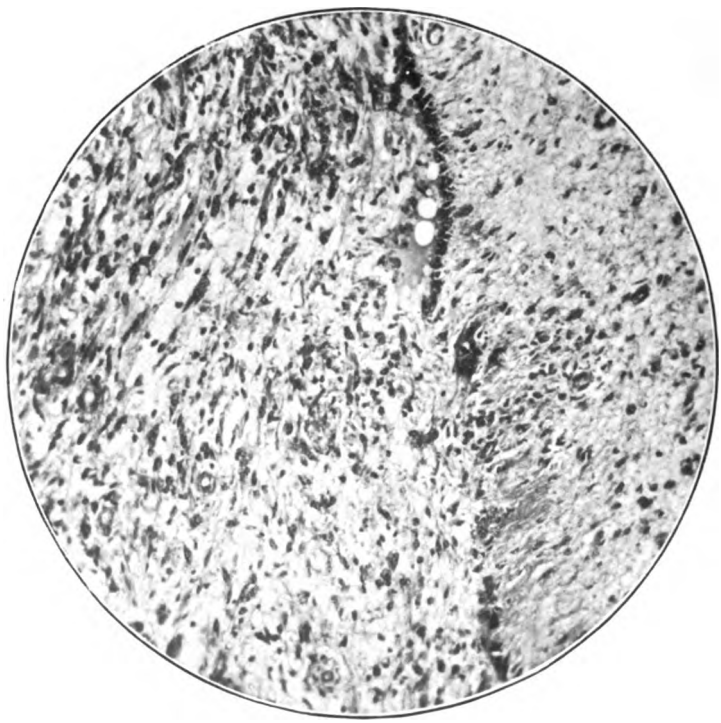

Fig. 4 (Case 1).—Photomicrograph of tumor showing remains of ependymal layer.

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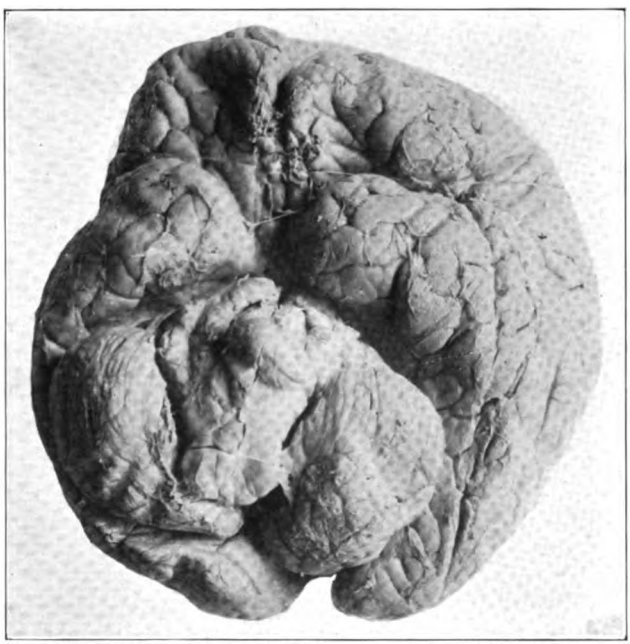

Fig. 5 (Case 2).—Base of brain showing tumor nearly encircling pons.

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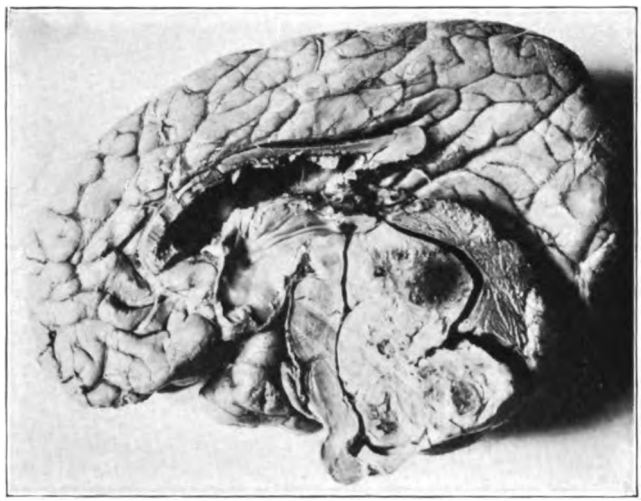

Fig. 6 (Case 2).—Median section of brain showing tumor filling fourth ventricle.

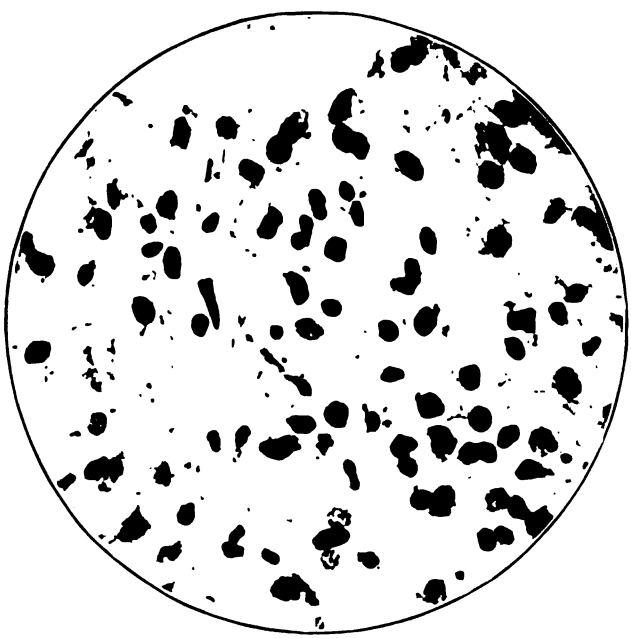

Fig. 7 (Case 2).—Photomicrograph showing glia fibers and cells.

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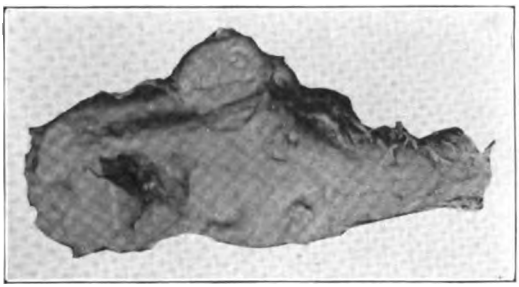

Fig. 8 (Case 3).—Tumor of fourth ventricle.

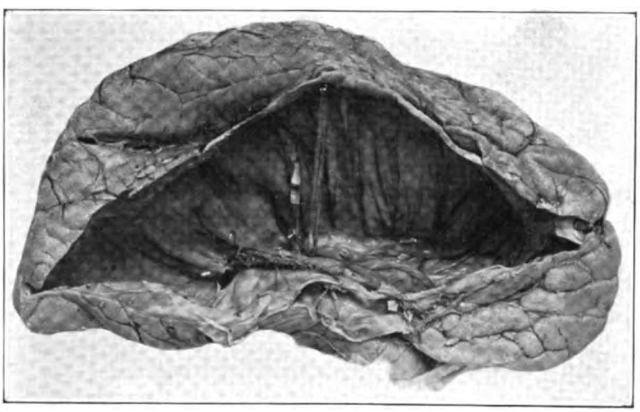

Fig. 9 (Case 3).—Median section of brain showing marked hydrocephalus.

Fig. 10 (Case 4).—Tumor in fourth ventricle.

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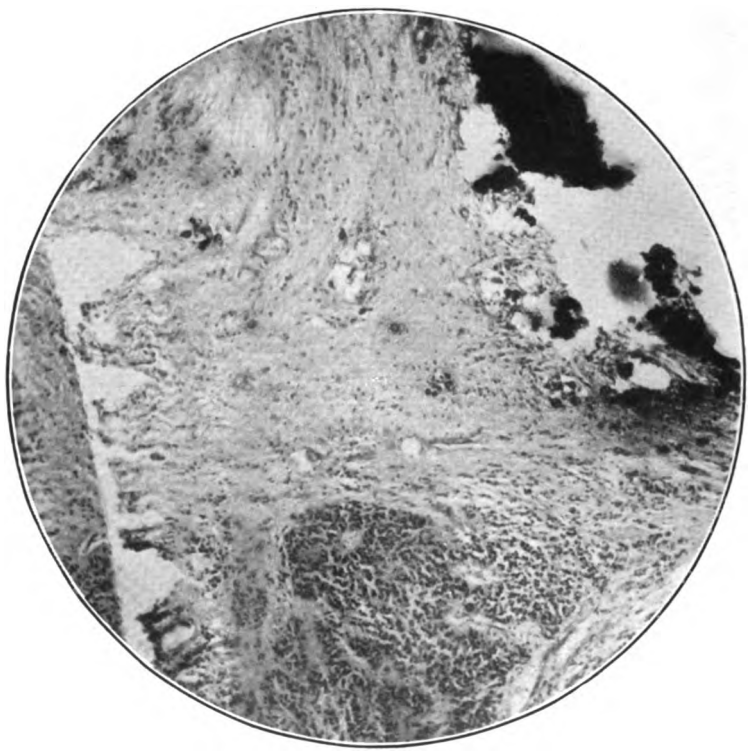

Fig. 11 (Case 4).—Photomicrograph showing deeply stained calcareous masses.

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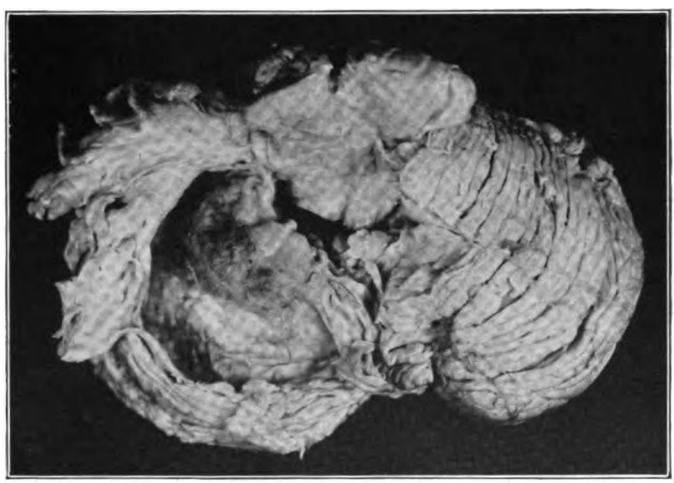

Fig. 12 (Case 5).—Tumor on left side of fourth ventricle.

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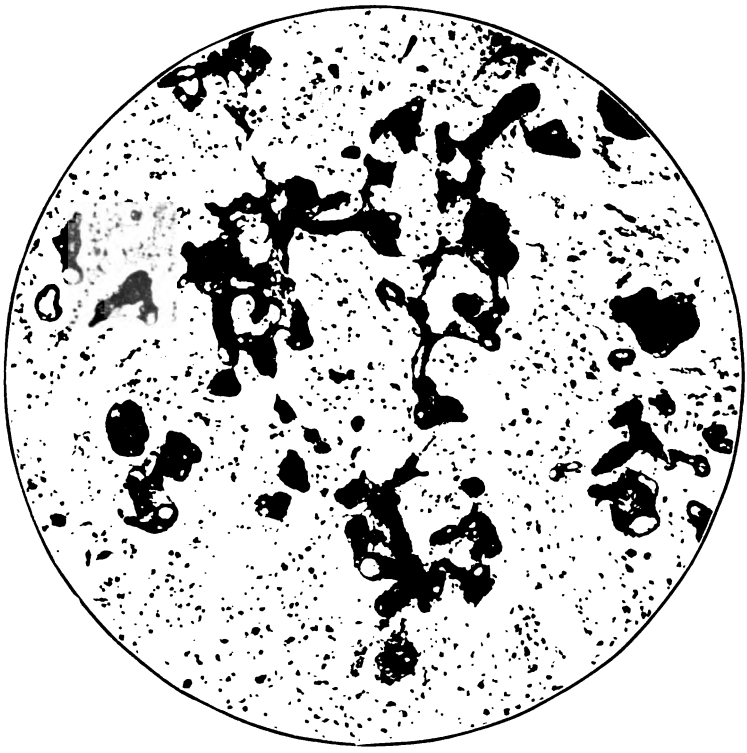

Fig. 13 (Case 5).—Photomicrograph of tumor showing hyaline transformation of vessels.

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he could stand up and walk when led by the hand. The headache was less severe, and vomiting had ceased. The spinal fluid was clear and showed a few lymphocytes, but no bacteria. A "weakly positive" Wassermann reaction was obtained with the spinal fluid, and the Lange gold test on this and three subsequent occasions gave a weakly positive reaction of syphilitic type, which may be represented as follows: 0 1 2 2 1 0 0 0 0 0 (maximum reaction would be 5). Mercurial treatment was now commenced and the patient continued to improve. March 2, a weakly positive Wassermann test with the blood was obtained. March 17, he walked well and had gained in weight. Lange test negative in all dilutions. No puncture was then made for a month, and by April 16 the headache had returned and marked ataxia had developed so he could not sit up in bed with the eyes closed. There was some relief after lumbar puncture. The Lange test was negative.

May 9: In spite of repeated lumbar punctures, the symptoms became more severe.

May 25: Frequent vomiting. Unable to sit up without swaying from side to side. Mind clear. Wassermann test with spinal fluid negative.

June 9: Died quite suddenly without any special symptoms having developed.

Necropsy.—After hardening in liquor formaldehydi the brain weighs 1,385 gm. The gyri are large and flattened, the sulci shallow. The cerebellar lobes at the base are seen to be separated by a light gray tumor mass which extends forward and upward so as to nearly surround the pons, only a space 8 mm. being left uncovered (Fig. 5). The width of the tumor between the cerebellar lobes is 3 cm., and it extends 4 cm. backward from the posterior border of the bulb. Figure 6 shows how the tumor fills and enormously expands the fourth ventricle. Hemorrhagic areas are seen in its substance. The pons and bulb are greatly flattened. The aqueduct is much dilated, the ventricles only moderately so. The tumor deeply indents the cerebellum on both sides, more so on the right, but does not grow into this organ or into the pons or bulb.

Histologic Examination.—The tumor shows characteristic structure of glioma, with typical rosette or fan-shaped arrangement of the cells about the vessels. Fine glia fibers are demonstrable by the Weigert glia stain (Fig. 7).

CASE 3.—*Clinical picture of cerebellar tumor. Death on operating table after exposure of dura over cerebellum. Small tumor in upper part of fourth ventricle. Marked hydrocephalus.*

A girl 5½ years old was examined, Oct. 8, 1912, in consultation with Dr. W. R. Parkes of Evanston. The child had been perfectly well up to the age of 4 years, when she had

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measles. Two or three months later she began to stumble in walking and would usually turn the eyes to one side. She had headache for a month at that time. The legs gradually grew worse. In November, 1911, a neurologist discovered optic neuritis, made a probable diagnosis of cerebellar tumor and advised operation, but his advice was disregarded. After a fall, four days before my examination, she began to vomit and became semistuporous.

Examination.—The head is unusually large, but this is probably not significant, as the six healthy brothers and sisters also have large heads. The patient is dull and drowsy, and does not at all cooperate in the examination. The pupils are equal, of medium width, and react normally. Bilateral papillitis of moderate degree, probably of long standing. No nystagmus; no facial paralysis. Ocular movements probably normal. All tendon reflexes increased. Both legs spastic, bilateral ankle clonus and Babinski sign. The upper abdominal reflexes were equal and normal, the lower doubtful. A diagnosis was made of tumor in the posterior fossa, probably cerebellar, and operation advised. October 15, a suboccipital operation was performed by Dr. Charles E. Kahlke, but the patient died before the dura was opened.

Through the courtesy of Dr. W. Henry Wilson, pathologist to Hahnemann Hospital, I later had an opportunity to examine the brain, which had been hardened in liquor formaldehydi and afterward weighed 820 gm. The upper part of the fourth ventricle is filled by a grayish, smooth, rounded tumor, attached to the floor for a distance of 2 cm. (Fig. 8). Its width is 15 mm., height 7 mm. Its inferior border is 2 cm. above the end of the ventricle. On making longitudinal section is appears as if the tumor at its upper end extends for at least 1 cm. into the pons. The brain shows very marked hydrocephalus (Fig. 9).

Histologic Examination.—The tumor is a glioma, rich in cells.

Longitudinal sections of the pons beneath the tumor show that the latter extends well into the pons substance. The border is diffuse. Pal-Weigert specimens show no persistent nerve fibers in the portion of the tumor growing into the pons.

A section of the upper part of the cervical cord, stained by the Pal-Weigert method, shows the posterior columns and the direct cerebellar tracts to be well preserved and deeply stained in comparison to the rest of the cord. The pyramidal and Gowers' tracts are decidedly degenerated.

CASE 4.—*Man of 58 years with symptoms of cardiovascular disease. Mental deterioration, delusions of persecution and delirious episode during last months of his life. Small sessile glioma in floor of fourth ventricle. External and internal hydrocephalus.*

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A cab driver 58 years old was admitted to the service of Dr. B. W. Sippy in the Presbyterian Hospital, Oct. 5, 1906. He had been a hard working and temperate man. He had acute articular rheumatism at 17 and at 30 years of age, and syphilis at 48. For several years he had occasionally had sudden momentary sensations that he was going to fall when walking, a sensation of constriction around the chest, and shortness of breath on exertion. There had been a general feeling of weakness for at least three years but no loss in weight, and the appetite had been good. While in the hospital he was suspicious of other patients, even claiming that they had robbed him and tried to murder him. His memory for recent events became greatly impaired. He gradually grew irritable and excitable, and three weeks after admittance became delirious, had involuntary discharges, and had to be restrained. After three days the delirium subsided and he returned to his previous mental state. He also developed a pleuritic effusion in the hospital and at first there were both a systolic and a presystolic murmur at the apex with irregular rhythm, but later the presystolic murmur disappeared and the systolic one became very faint. The eye grounds were examined twice and found to be normal. The blood pressure at first was 190, later 104. The urine frequently contained albumin and hyaline and granular casts. The hospital record is silent as to any symptoms and findings on the part of the nervous system other than those mentioned. The patient died, Feb. 8, 1907.

Necropsy.—At the necropsy the following anatomic diagnosis was recorded: arteriosclerosis; thrombosis of left iliac artery; retrograde thrombus formation in the aorta; edema of lungs, fibrinous pericarditis; hypertrophy of heart; chronic mitral and aortic endocarditis; hypertrophy of prostate and bladder; multiple rectal ulcers; tumor of fourth ventricle; external and internal hydrocephalus; atrophy of brain.

On opening the fourth ventricle a growth was seen attached to its floor and extending into the lumen of the ventricle but not reaching its roof. It was 1.5 cm. long, 1.7 cm. wide, and 1.8 cm. in height (Fig. 10). The protocol described the brain as "soft and tense to bursting." There was a large amount of subdural fluid, and the pia-arachnoid was very edematous. The ventricles were dilated.

Histologic Examination.—The tumor shows the structure of ependymal glioma, with some areas consisting mostly of fine fibers while in others cells predominate. In the center of the tumor small areas of calcification, as well as hyaline concretions are seen (Fig. 11).

CASE 5.—*Man, aged 38 at death. For several years attacks of headache, dizziness and vomiting. Early relative weakness in left side. Mild mental deterioration and symptoms of car-*

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diac and renal disease. Death from hypostatic pneumonia. Sessile tumor in floor of fourth ventricle.[5]

A business man, married, 37 years old, was first seen by the writer, Nov. 15, 1912. There was some suspicion of syphilis in the father. No brothers or sisters. No previous illnesses of importance. Venereal disease denied.

Present Illness.—The onset was very gradual. About five years before death (1909) he began having brief attacks of nausea and vertigo, later associated with headache of increasing severity, lasting in the beginning a few hours only, later several days. From about June, 1911, more or less vertigo was experienced every day, worse in the mornings and after heavy meals, and especially when stooping. Vomiting occurred with severe attacks of headache. He began losing flesh in 1910, but when examined by Dr. Portis in November, 1911, he still weighed 195 pounds; one year later, 152 pounds. Dr. Portis found gastric hyperacidity and low blood pressure, (96 to 108). In 1911 he began complaining of clumsiness and lack of control of the movements of the left hand and arm, especially annoying when shaving or driving his automobile. A tendency to drag the left foot and to stagger to the left was also noted at this time. When first seen by Dr. Davis in March, 1912, physical examination was practically negative. The Wassermann test with the blood was made several times, generally negative, once or twice "doubtful." Later in 1912 the weakness in the left side increased and he showed a suspicious overconfidence in his business ability. In October of the same year he became involved in business failure and legal prosecution, and showed apparent rapid increase in mental deterioration which, however, possibly to some extent was simulated.

Examination by the writer, Nov. 15, 1912: Patient carries himself like an old man and talks slowly and feebly. Memory probably somewhat impaired. Decided relative weakness in left arm and leg, latter slightly atrophic. Both knee and ankle jerks brisk, left more so. Pulse 68. No other findings.

Dec. 8, 1912, reexamined. Looks better, Has had headache every day, worse when sitting up. Less difference in reflexes on right side and left side, but left leg appears weaker. Blood pressure 100, pulse 64. Wassermann test with blood and spinal fluid negative. The latter shows no increase in cells or globulin. Lange colloidal gold test negative. Ophthalmoscopic examination negative. Attacks of severe headache, with vomiting, recurred frequently. The patient was last seen by me in 1913. Dr. Davis kindly informed me that he was under his continued observation from the fall of 1913 until his death in April, 1914. The weakness and atrophy of

5. This patient was only under my observation for a period of a few months. For a large part of the clinical history I am indebted to Drs. Achilles Davis and Milton Portis, and for the necropsy notes and opportunity to examine the brain to Dr. F. Robert Zeit.

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the left limbs increased. The eye grounds remained normal, but headache, vomiting and dizziness remained prominent symptoms. Albumin and casts appeared in the urine. Repeated examinations of the spinal fluid showed nothing abnormal. No sensory disturbance developed. A few days before death he developed hypostatic pneumonia which determined the fatal issue.

Necropsy.—This was held by Dr. F. Robert Zeit, who kindly placed his notes and the gross and microscopic specimens at my disposal. The following anatomic diagnosis was made by Dr. Zeit: Ependymal glioma of fourth ventricle. Internal hydrocephalus. Edema of brain. Chronic parenchymatous nephritis (large, red kidney). Hypertrophy of heart. Brown induration of lungs. Terminal hypostatic pneumonia. Healed tubercle in apex of left lung. Chronic granular gastritis and hemorrhagic acute duodenitis.

The specimen further examined by me consisted of the cerebellum, pons and medulla. The cerebellum had been opened from above so as to expose the fourth ventricle. This is greatly distended, and on the left side it forms a cavity occupying the greater part of the cerebellar hemisphere. To the left of the median line the ventricle is the seat of a sessile, grayish-red growth measuring 3.3 cm. in length, 3.5 cm. transversely, and projecting 8 mm. into the ventricle (Fig. 12). The cavity extends 2 cm. to the left of the tumor margin and has a smooth lining. The cerebellar substance covering the dilated ventricle on the left side is only 3 to 5 mm. in thickness. The under surface of the cerebellum shows marked molding at the edge of the foramen magnum.

The other gross findings are summarized in the anatomic diagnosis. It was particularly noted that nothing suggestive of a syphilitic lesion was found in the body, that there was very little sclerosis of the aorta, and that the vessels at the base of the brain had soft and thin walls. The skull and dura showed no changes. The convolutions of the brain were flattened, and the lateral and third ventricles were considerably dilated. Each kidney weighed 230 gm., and the capsules were adherent. The heart weighed 330 gm.

Histologic Examination.—The structure is that of a somewhat degenerated ependymal glioma. Numerous hyaline concretions are seen (Fig. 13), mostly derived from obliterated vessels.

A Pal-Weigert specimen of the bulb shows considerable degeneration of the dorsal portion of the pyramids, adjoining the olives, while their ventral (superficial) portions are well preserved. Still more marked degeneration is found in the raphe and in the interolivary fibers. This portion of the bulb is seen in hematoxylin-eosin and Van Gieson preparations to be thickly set with pale blue, hyaline, round bodies.

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CASE 6.—*Boy 5 years old. Onset with headache, then adiposity, unsteadiness, papillitis and failing vision. Genitalia said to have become smaller. Later blindness, two convulsive seisures, marked general weakness. Decompressive operation, hyperpyrexia, death. Tumor filling fourth ventricle.*

A boy 5 years old was referred by Dr. Wright C. Williams of Peoria, Feb. 16, 1914. The patient was the sixth of eight children, the others being well. In the summer of 1913 he began to suffer from headache; later he grew fat and the genitals grew smaller. In the beginning of December he was brought to Dr. Williams on account of headache and a tendency to stumble. There was a slight papillitis, especially in the right eye, and vision was considerably reduced. It failed rapidly, and he became completely blind about the first of January. Late in December there were two convulsive seizures three days apart. The Wassermann test was negative with both blood and spinal fluid, and two examinations of the latter for cells were negative. Urine negative.

Examination.—Fat child with flabby musculature. Penis small; both testicles in the scrotum. Patient very weak; cannot walk or sit up unaided or hold the head erect for any length of time. Totally blind. Pupils equal and dilated. Constant rotary nystagmoid movements drawing the eyeballs upward and to the left. The movement to the left is rapid, the return slow. The eyes wink with the beginning of each nystagmoid movement. Dr. E. V. L. Brown made the following note: "Right and left optic nerve atrophy, secondary to marked choked disks; nerve head swollen (right plus 2); scleral and pigment rings not seen; retinae not involved; vessels narrow; pupils react to light only with light directly in front." Hearing normal. Apparently slight paresis of right side of face and right arm. Knee jerks absent; ankle jerks slightly increased, right more so; bilateral Babinski sign. The patient could draw the knees up but could not raise the knees from the bed. Probably no sensory disturbance . Abdominal reflexes not obtained at first, but later weak response was obtained on both sides. Soft, blowing heart murmur at the base. Roentgen-ray examination showed normal sella turcica. Blood examination: reds, 4,940,000; whites, 11,000; hemoglobin, 98. Urine normal.

February 25: Right subtemporal decompression made by Dr. Dean Lewis. The brain pressure was very high. Soon afterward the arms and later the entire body twitched continually. The temperature rose to 108.6 F. before death, eighteen hours after the operation.

Necropsy.—The brain weighed 1,170 gm. Marked downward bulging of the third ventricle was noted. The hypophysis was normal. The fourth ventricle was filled by a soft tumor the greatest dimension of which was 5 cm. transversely and 3.8 cm. vertically. The tumor was connected with the

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pons. Unfortunately, I missed the opportunity to examine the hardened and sectioned brain, and failed to learn to what extent the pons was actually invaded. All of the ventricles were considerably distended. The tumor tissue examined microscopically was said to be made up of masses of darkly staining small round cells and to show considerable necrosis and in places small hemorrhages.

The histologic diagnosis was small round-cell sarcoma. (I have not had an opportunity to see the specimens, and know from experience that sarcoma and cellular glioma are frequently confused.)

THERAPEUTIC REMARKS

Of these six patients three were operated on, all with disastrous results. In Cases 1 and 6 subtemporal decompression was followed by death in thirty and eighteen hours, respectively. In Case 3 a suboccipital operation resulted in death on the table before the dura was opened. In Case 2 repeated lumbar puncture apparently gave temporary relief at times, but it naturally would more often be found to aggravate the symptoms. Any sudden interference with the intracranial pressure condition appears to be dangerous, and this applies to the three methods used in the present cases. The danger of death from the application of any of the usual cerebral decompressive methods, the practical impossibility of making a clinical diagnosis of intraventricular tumor, and the difficulty of access to these ventricles all conspire to place tumors of the third and fourth ventricles among inoperable brain tumors in the minds of most neurologists and surgeons. However, the writer must agree with Professor Anton[6] of Halle that operative removal of tumors of the fourth ventricle is not theoretically impossible. Some of them arise from the choroid plexus and are not attached to the walls, and others are only attached above, to the cerebellar vermes. It would, therefore, seem possible in such cases to enter the ventricle through the vermes, or below, and possibly remove the tumor, provided the pressure in the posterior fossa

6. Anton: Arch. f. Psychiat., 1911, xlviii, 523.

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was not too high at the time of operation. In order to obtain a favorable pressure condition, Anton recommends operating at a period of relative freedom from headache and other pressure symptoms, and to perform a preliminary callosal puncture or puncture of one of the lateral ventricles. The only practical suggestion to offer is this one, that if symptoms strongly point to posterior fossa tumor and nothing is found in the cerebellar hemispheres, then the surgeon may go a step further and also explore the fourth ventricle, at the same sitting, if a preliminary callosal puncture has been made, or else after an interval of a few days.

30 North Michigan Avenue.

ABSTRACT OF DISCUSSION

Dr. Theodore H. Weisenburg, Philadelphia: Years ago I wrote a paper on this subject in which I reviewed the cases of tumor of the third ventricle reported in the literature. I wrote this paper because I had had three cases of tumor of the third ventricle in every one of which diagnosis of tumor of the cerebellum was made. On investigation I found that of the forty cases reported, not a single one had ever been diagnosed properly during life. I then tried to establish a symptom complex. Most cases of tumor of the third ventricle cause cerebellar asynergy; they all have internal hydrocephalus, with a para-paresis of all four limbs with the accompanying increase of reflexes. With this there is nearly always paresis of associated ocular movement upward. This symptom group would seem to be especially indicative of tumor of the third ventricle, and Dr. Bassoe's case would seem to confirm this. I believe that we can make a diagnosis of tumor of the third ventricle in life, for I have been able to do so. Tumors of the fourth ventricle are quite difficult to diagnose. Not in a single case, I believe, has a diagnosis been made in life. In most of these cases the diagnosis has been tumor of the cerebellum. Dr. Mills and I tried to show that there is such a thing as cerebellar localization. In cerebellar localization the trouble has been that there is usually no attempt to make a diagnosis of tumors in the separate parts of the cerebellum. Recently I have had two cases in which from our localization I was enabled to make an exact diagnosis. In one a tumor of the superior portion of the right cerebellum was diagnosed and operated, with removal of the tumor, and the child is now living and perfectly well. In another case, that of a man, we diagnosed tumor of the left lobe of the

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cerebellum, and found and removed the tumor. Localization in the cerebellum should be advanced, that is, we should not be content to make diagnosis of tumor of the cerebellum; but should be able to say that a lesion is located in a certain and definite part of the cerebellum.

Dr. Tom A. Williams, Washington, D. C.: In tumors of the third ventricle there is another sign to be found which was not brought out by Dr. Bassoe in his case report, and I do not remember whether it was brought out by Dr. Weisenburg in his original communication. The sign is one that was shown in a case we had in Washington in 1907 which was diagnosed during life and in which we had a postmortem. There were photographs in the case. Diagnosis was made of tumor in the third ventricle or of the corpora quadrigemina, involving both sides, and it was made by reason of several findings: First, because of complete ophthalmoplegia; second, from the eye grounds because of the marked choking of the disk in conjunction with the extreme rapidity of the onset; and, third, on account of the decerebrate attitude of the patient. This attitude is one that was, I believe, first described in connection with physiologic experiments, and consists of the quadrupedal progression reaction, namely: Suppose you scratch the right arm of the subject, there will immediately be, let us say, flexion of the right arm, and at the same time there will occur flexion of the counter leg, along with extension of the contra-lateral arm and extension of the homolateral leg; that is to say, the attitude and movement of quadrupedal progression. This is due to impulses from the spinal centers when separated from cerebral control. On that ground mainly the diagnosis in this case was made. And photographs show the child's position after stimulation of one area in various attitudes, as described, in that position, which is the position of the dog's progression. Hughlings Jackson published a case showing this attitude. I do not remember whether that was a case of this particular lesion, neither do I know whether a necropsy was obtained. The sign may depend on rapidity of onset, because it is possible that, in the gradual progress of a unilateral tumor slowly invading the third ventricle, gradual adaptation may prevent this particular syndrome or the patient may die before it develops.

Dr. Harold N. Moyer, Chicago: I saw the fifth case described by Dr. Bassoe on one occasion but the opportunity for examining was none of the best. In this case there was a striking clinical resemblance to paretic dementia. The symptoms would become very pronounced, then subside, and his conduct in the periods of euphoria, which were marked, strongly suggested paresis. He was distinctly paretic on the left side and persistently leaned forward and to the left; he would straighten up, but would

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soon automatically resume this position. I expressed the opinion that he had a brain tumor but of its location I had no idea. I certainly never suspected that it was a subtentorial growth. Dr. Bassoe says the eyegrounds were normal. At the time I examined him it seemed to me that the left disk was somewhat altered but not significantly so.

Dr. Lawson G. Lowrey, Hathorne, Mass.: About a year and a half ago I made a necropsy on a case presenting a tumor presumably arising in the third ventricle and extending from there into the lateral ventricle. For two years the patient had been in Danvers hospital with a diagnosis of paresis. The symptoms were largely those of paresis, the onset had been rather gradual, the individual was dull, extremely indifferent and had demented very rapidly, with a doubtful blood Wassermann and no satisfactory spinal fluid examination. This tumor, too, was a glioma. So far as any exact examination that had ever been made showed there were no symptoms that might be referred to a tumor.

Dr. A. L. Skoog, Kansas City: While these tumors of the third and fourth ventricles are difficult to diagnose, yet frequently you can suspect localization. Probably quite a number of errors have been made on account of not giving sufficient consideration to the chronology of the case. We know that in cases exhibiting these various symptoms, and particularly those referrable to the cerebellum and the medullary centers, and also those due to the secondary internal hydrocephalus, that there is an onset of the symptoms at different periods. I believe we should lay emphasis on carefully noting just when each symptom begins. In this respect a careful history is, of course, important. There are case histories that are accurate and others that are not.

Dr. Peter Bassoe, Chicago: I must repeat what I said at the outset; that the symptoms in these tumors are caused either by pressure or invasion of neighboring organs, or by hydrocephalus. Dr. Skoog says, by a study of a sequence of symptoms we may form some opinion as to where the tumor originated. In the case of the fourth ventricle, if a tumor starts from the choroid plexus, there would necessarily not be symptoms until a certain amount of hydrocephalus was produced. If the tumor starts from the floor you may get symptoms on the part of the cranial nerves, or if it starts from the vermes you may have early localized cerebellar symptoms. In the case of the third ventricle, quite necessarily the symptoms are those of hydrocephalus. We can hardly speak of any focal symptoms there unless the tumor has grown from the ependyma into the brain structure, or has started in some other structure and extended into the ventricle. In my only case of tumor of the third ventricle, the patient had a typical cerebellar ataxia. The very early loss of conjugate movements and early blind-

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ness, and the quite precocious development of hair and of the genital organs led us to suspect a tumor in the general vicinity of the pineal gland, or corpora quadrigemina, or in the third ventricle, but we did not venture to say in which of these structures the tumor might have started.

In regard to the diagnosis between tumors of the fourth ventricle and tumors starting in the cerebellum, this is extremely difficult. But if in a case with ordinary cerebellar tumor picture there is marked variation in the subjective feeling of the patient on changing position, together with quite a marked increase in cells and globulin of the spinal fluid, then I would be suspicious of a tumor in the fourth ventricle. In the case of which Dr. Moyer speaks, there was no change in the disks that I could make out, but I did not examine the patient during the last year of his life. I was told by three or four of the men who saw the case that they had never noted any change in the disks, but the patient was not under the observation of any one man during the whole period.

There is one aspect that has not been touched up here, and that is the possibility of any surgical treatment in these cases. In my three operated patients the surgical results were notoriously bad; one died on the table and the other two did not rally. Could we have done any better for those patients? It is only within a year that Professor Anton of Halle has written a paper seriously proposing operative procedure in cases of tumor of the fourth ventricle, providing it arises from the roof or choroid plexus. He says that at least half the tumors arise from the roof. By first doing a callosal puncture or puncture of the lateral ventricle, thus relieving the pressure from above, the suboccipital opening can be made with less shock. He believes it might be possible to go through the vermis, which usually has been gradually thinned out by the pressure, as was the case in some of my cases, or that we might go through the foramen Magendie, and remove the tumor. About ten years ago it was suggested by Henneberg that cysticercus cysts of the fourth ventricle might be removed in that way.

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A FURTHER NOTE ON THE DIAGNOSTIC VALUE OF RETROBULBAR NEURITIS IN EXPANDING LESIONS OF THE FRONTAL LOBES

WITH A REPORT OF THIS SYNDROME IN A CASE OF ANEURYSM OF THE RIGHT INTERNAL CAROTID ARTERY

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Difficulty in the diagnosis of cerebral lesions varies directly with the degree of physiologic latency possessed by the affected brain area.

Again, even when tissue subserving active function becomes irritated or destroyed by disease, our difficulty in decision as to the part involved depends largely on the degree of objectivity in the phenomena produced.

The vertigo accompanying cysticercus of the fourth ventricle eludes the descriptive powers of the majority of patients, nor are the uncinate fits associated with irritative processes in the hippocampal gyri more easily portrayed: in consequence of the difficulty in describing concretely such subjective states, occurrences of the most precise character are apt to be misinterpreted by even alert observers. In the recognition of disease affecting the frontal lobes there is ample illustration of the truth of these general propositions. These gyri are not only highly latent, but such exiguous symptomatology as has been ascribed to them consists mainly of certain ill-defined abnormalities of mentality and conduct.

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It is, therefore, proposed in this short communication again[1] to draw attention to a syndrome referable to the inferior frontal areas, a clinical picture also which is capable of such objective investigation as to facilitate decisively the diagnosis of pressure-producing lesions of these parts.

The sign of which I wish to speak is the occurrence of a true retrobulbar neuritis, with the formation of a central scotoma and primary optic atrophy on the side of the lesion together (if pressure be sufficiently great) with ipsolateral anosmia and papilledema in the opposite eye. The mode of production of this situation may be illustrated by the case of a woman, aged 53, kindly referred to me by Dr. John Shannon in February of this year. This patient complained only of blindness in the right and decrease of vision in the left eye. The history was as follows:

All her life she had been apparently strong and of good physique and health; she had borne three children, all of whom were healthy. There had been no miscarriages. In September, 1915, without any previous premonitory symptoms, she found that she was not seeing clearly with the right eye. This symptom was definite from the first and progressed from week to week. An examination at that time showed, first, that there was an enlargement of the right blind spot and, later, the definite formation of a small central scotoma, complete for form, and a larger scotoma encircling the other, complete only for color. This central visual defect became, as the weeks passed, more pronounced until, at the end of two months, the patient was able only to see moving objects in the periphery of the right visual field. Most properly her frontal and sphenoidal sinuses fell under suspicion. Roentgenograms were taken which failed to demonstrate any abnormality of these parts. However, lest a mistake should have been made in the radiographic deductions, an exploration of the sinuses was performed, with negative results. There were no other subjective symptoms at that time, but the reaction of the right pupil was diminished in extent; the pupil reacted to light quickly, though with small amplitude, but speedily rebounded, the degree of illumination having been maintained the while. This phenomenon has been called the retrobulbar pupillary reaction, in that it occurs apparently exclusively

1. Kennedy, Foster: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses of the Frontal Lobes, Am. Jour. Med. Sc., September, 1911.

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in conjunction with retrobulbar neuritis. The existence of this latter condition was further demonstrated by the fact that the right optic nerve, as seen by the ophthalmoscope, showed a distinct pallor in its outer part. The patient remained well with the exception of occasional very slight headache.

This headache was adjudged to be the result of the various sinus operations and so excited but little comment. The patient never had experienced headache throughout the whole of her life. In February, 1916, when shopping, she noticed slight dimness of vision affecting the left eye. A perimetric examination, carried out that day by Dr. Shannon, revealed an enlargement of the left blind spot, which in the next three weeks increased in size to form a central scotoma in the left visual field, in a manner precisely the same as had occurred on the right side six months previously.

The diagnosis made by various observers at that time was that the patient apparently was the subject of a so-called von Leber's optic atrophy. It may be pointed out here that this group of atrophies is indeterminate and vague, and that the etiologic factor is only presumed to be obscurely toxic. When she came under observation in February, however, it appeared certain that a toxic factor would not operate by attacking and destroying a single optic nerve and then resume its activity on the other six months later. A disseminated toxin has symmetrical and synchronous results.

A careful examination then revealed the fact that previous to her attack of September, 1915, she had had three transient attacks of double vision, and that after that date she had been troubled with frequent transient numbnesses in both hands and feet, these being rather more evident on the left side. Neurologic examination then showed the condition of the right pupil to be as already described. A similar state affecting the left pupil was incipient. A very slight haziness was made out on the upper nasal margins of both disks. The physiologic pit in each was clear, the lamina cribrosa evident, and the blood vessels undisturbed.

Her sense of smell was acute in both nostrils. No nystagmus, strabismus or diplopia. The hearing was good. The area of distribution of the fifth nerve was unaffected. Repeated examination gave rise to the suspicion that the lower part of the left side of the face was less strong than its fellow.

There was a well-marked fine tremor in the right hand when held outstretched. This was quite constant, was diminished by purposeful movement, and never affected the left hand. It was thought there was a little weakness in the left grasp.

The deep reflexes on the left side were rather doubtfully greater than those on the right. The abdominal reflexes at the beginning of the examination were on each side explosively brisk, but repeated stimulation, first of the right side

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then of the left, maintained over a period of seven minutes, produced an important and interesting situation; at the end of that time the abdominal reflexes on the right side were almost as active as at the beginning of the experiment, whereas those on the left side had completely disappeared.

The plantar reflex on the left side was very sluggish and of equivocal type, that on the right was invariably flexor. These facts led to the postulate that the motor fibers subserving the left side of the body were slightly deteriorated in function. A cause capable of producing the blindness and the very minute degree of left-sided hemiparesis would necessarily be one pressing downward on the optic nerves and upward on the motor fibers coming from the right side of the brain.

Furthermore, the lesion would have to be of an expanding character and so capable of exerting pressure in different directions. In one of the Roentgen-ray plates a slight shadow was seen in the frontal region, a shadow which seemed to support the theory that the patient was the subject of a tumor lying at the base of the right frontal lobe. The fact that the tumor was sufficiently large to reach from the right to the left optic nerve and that six months were required for symptoms to be developed on the left side after the affection of the right, together with the entire absence of the generalized symptomatology associated with the development of brain neoplasm—all these considerations pointed to the benignity of the growth. It appeared that, in all probability, the tumor would turn out to be an endothelioma, a type of neoplasm of slow growth, frequently sufficiently dense in structure to give evidence of its presence by radiograph, and, by reason of its very gradual enlargement, capable of becoming of great size without at the same time producing marked cerebral symptoms. The brain tissue in such cases is given sufficient time gradually to accommodate itself to the encroachments of the foreign body and so there is produced no more than a very slight deterioration of function.

One may point out here the fact that the patient was able to smell distinctly in each nostril, a circumstance which occasioned the observer no little surprise. A growth sufficiently large to compress both optic nerves might have been considered capable of compressing both olfactory tracts. The other circumstances of the case, however, so strongly pointed to the existence of tumor formation in the situation mentioned, that operation was decided on and carried out. Dr. Elsberg exposed and elevated as far as possible the right frontal lobe and succeeded in demonstrating a large periphery of the sublying bone. On the inner surface, near the falx, some pinkish-gray, gelatinous-looking tissue was seen, which was thought by us to be the outlying part of a glioma, the central origin of which could not be brought into view.

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A decompression operation was consequently carried out without, fortunately enough, any attempt being made to remove the growth. The patient recovered excellently from the immediate effects of the operation and remained feeling perfectly well for some two and a half months, with the exception of some doubtful further reduction of vision in the left eye. At the end of this time, however, it was discovered that complete anosmia had occurred, which gave rise to the belief that the tumor had increased very considerably in size. A week after this examination the patient fainted one morning, there was a slight convulsion, chiefly on the left side of the body, the coma deepened and death occurred six hours later.

An examination of the brain revealed a ruptured aneurysm of the right internal carotid artery about the size of a small walnut and of a solid consistency, owing to contained fibrinous matter. This aneurysm caused marked pressure on the right optic nerve, just in front of the chiasma, and, to a lesser degree, on the left optic nerve also. Upward and to the right it had succeeded in compressing descending fibers from the right motor area with some deformation of the anterior part of the right caudate nucleus. The anterior horn of the right lateral ventricle was considerably larger than its fellow.

This case is shown for two reasons: first, because of the remarkable rarity of aneurysms of a right internal carotid artery; and second, and chiefly, because, as has been said, its symptomatology offers a very important clue to the diagnosis of expanding lesions in the subfrontal area. Papilledema is now generally believed to be the result of the forcing of fluid out of the cerebrospinal pond down the vaginal sheath of the optic nerve to the nerve head. There it accumulates and is seen by the ophthalmoscope as a localized papillary swelling combined with venous tortuosity. Such is the mode of production of papilledema brought about by the presence of an expanding lesion anywhere in the cranial cavity, so long as this lesion does not take origin from the beginning directly in contiguity with the optic nerves themselves. When this latter phenomenon occurs, a completely different series of events takes place. Instead of fluid being forced down the vaginal sheath there is an obstruction on the vaginal sheath preventing the possibility of such an

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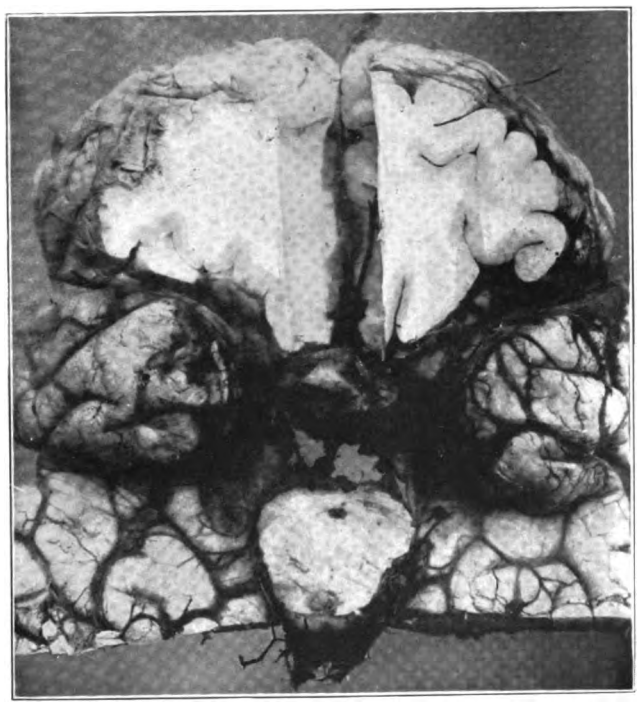

Fig. 1.—Inferior aspect of brain, showing aneurysm of right internal carotid artery. The optic nerves have been removed and would lie on the aneurysm, between it and the observer.

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Fig. 2.—Section through brain showing upper aspect of aneurysm. Notice compression of right caudate nucleus and capsule, with enlargement of anterior horn of right lateral ventricle.

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occurrence. Also the growth, by its pressure on the nerves, causes degeneration to occur directly in the nerve itself. The order in which the fibers of the optic nerve become affected is constant.

We are aware of the existence of varying degrees of physiologic delicacy in the optic nerve bundles, according to the retinal region in which they originate; parallel degrees of susceptibility to trauma would likewise appear to occur in the same fibers. That is to say, the macular bundle which subserves central vision, while not placed peripherally in the nerve, and so not exposed to direct pressure from without, owing to its aforesaid greater vulnerability will always degenerate earlier than the fibers more directly compressed. The result to the patient will be a rapid loss in the acuity of vision and the formation of a definite central scotoma. In the majority of cases this will be accompanied by deterioration of function in the ipsolateral olfactory bulb. This was spared until late in the disease, in the present instance owing to the posterior situation of the lesion.

The diagnosis of aneurysm in this case was, of course, quite unsuspected during the patient's lifetime. The presence of syphilis was entirely excluded by the negative history and annectant circumstances, by repeated examinations of the blood by the Wassermann reaction, all of which were entirely negative, and by the existence of a cerebrospinal fluid normal in all respects with the exception of an increased globulin content, a finding which only served the more to strengthen one's belief in the presence of new growth.

However, the signs which have been here discussed were sufficient to make plain the exact situation of an expanding lesion within the skull cavity. It is submitted that the recognition of this syndrome may do something to elucidate diagnosis in certain obscure cases of frontal neoplasm and, perhaps, at the same time serve in a measure to separate a group of cases from the great generic class of toxic amblyopias.

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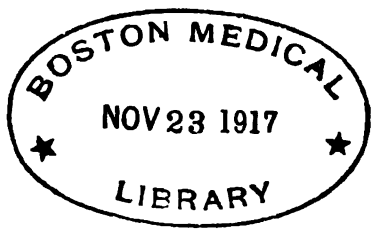
ABSTRACT OF DISCUSSION

Dr. Francis R. Fry, St. Louis: I think it was Marcus Gunn, an English ophthalmologist, who first affirmed that the "rebounding pupil," the pupil described by Dr. Kennedy, was pathognomonic of retrobulbar neuritis. I think, however, that in so considering it there might be some confusion, because we certainly see a pupil behaving in this way, sometimes unilaterally, where there is no retrobulbar neuritis. And it might only be accepted, I think, as of affirmative value, when the fact of neuritis is otherwise suspected.

Dr. Foster Kennedy, New York: I assure Dr. Fry that I would not think of diagnosing lesions of the frontal lobes on the pupillary reaction alone. This would only be of value, of course, in corroboration of other phenomena.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WEAKNESS OF THE SOFT PALATE AND OF THE TONGUE A CONSTANT SYMPTOM IN HEMIPLEGIA

P. DE LONG, M.D.
AND
T. H. WEISENBURG, M.D.
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The extent of the paralysis on one side of the body and the escape of certain movements in hemiplegia have always been interesting problems. The latter has always been explained by Broadbent's hypothesis that those muscles which act bilaterally are represented on both sides of the brain, and therefore a unilateral lesion at most could only produce a temporary paralysis.

At first it was supposed that most of the so-called bilaterally acting movements escaped in a cerebral hemiplegia. In this were included wrinkling of the forehead, moving of the eyeballs, the muscles involved in eating, talking, swallowing, chewing, and the muscles of respiration and of the abdomen. Gradually, however, this view has been modified until at present there are some neurologists who hold the opinion that in a cerebral hemiplegia there is a partial implication in all of the so-called bilaterally acting movements.

Weakness in the upper facial distribution in cerebral hemiplegia has been frequently noted. Mirallie,[1] for example, is of the opinion that in every case in which there is weakness of the lower facial muscles the upper facial is also affected, but to a less degree, while von Monakow[2] states that careful examination of the paralyzed side will reveal a delay in the closing of the eye

1. Mirallie: Arch. de neurol., 1899, p. 1 et seq.
2. Von Monakow: Gehirnpathologie, p. 288.

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and the wrinkling of the forehead, and that in fact every upper facial muscle has suffered a certain slight and transitory loss of power. We are able to add a rather interesting observation in regard to this point, for it can be accepted at once that stimulative experiments of the cortical facial center should give contributory results. One of us[3] for a long time has been studying nervous cases by the use of moving pictures. In a case of jacksonian convulsions in which the twitchings always started in the right corner of the mouth and extended to the right side of the face and upper limb, the upper facial distribution was alone involved in the beginning, and it was only later on in the convulsion that the left eyebrow also took part in the movement. In the same picture it has also seemed to us as if the right eyeball was jerked to the right much more than the left eyeball. It is to be remembered that the irritation was on the left side.

Mirallie and Desclaux[4] have attempted to show that in every case of cerebral hemiplegia in which the face is involved, all the muscles of the eye on the paralyzed side are affected to a greater or less degree. This view, however, has not been generally accepted. For example, S. A. K. Wilson,[5] comes to the opposite conclusion, and holds ocular movements are absolute in their bilateral representation and innervation.

The masseters, temporals and pterygoid muscles have, of course, been frequently found to be involved on the paralyzed side. The weakness of the tongue in hemiplegia is very well known. The weakness of the soft palate is mentioned, but the extent of its involvement is not of common knowledge. It has been usually accepted that the larynx is not implicated in hemiplegia, and yet in a careful survey of the literature we have been unable to find a really satisfactory work which proves this point.

3. Weisenburg, T. H.: Moving Picture Illustrations in Medicine, THE JOURNAL A. M. A., Dec. 28, 1912, p. 2310.
4. Mirallie and Desclaux: Rev. neurol., 1903, p. 649. Desclaux: Thèse de Paris, 1903.
5. Wilson, S. A. K.: Rev. Neurol. and Psychiat., 1904, p. 265.

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Of the muscles of the neck, chest and abdomen, it is, of course, known that the platysma and abdominal muscles are weak in hemiplegia and that the muscles of respiration are undoubtedly affected.

The present investigation is concerned with the extent of the paralysis of the palate and tongue in cerebral hemiplegia.

Our results, in brief, show that in practically every hemiplegia in which there is facial involvement, the soft palate is either totally or partially weak on the paralyzed side. In fact, so impressed have we been with the consistency of this symptom that we are loath to accept the presence of a hemiplegia in the absence of a weakness of the palate on the paralyzed side. This is equally true of the tongue; but the weakness of this organ is not so readily shown as that of the palate, and therefore as a constant symptom it does not have the importance of the former.

We investigated in round numbers 100 cases of hemiplegia, all of these being either in the nervous wards or the out-wards of the Philadelphia General Hospital. Besides, we studied a number of cases of diplegia and pseudobulbar palsy. Of the hemiplegics, in every instance in which we obtained a good view, there was found to be weakness of the soft palate.

In a normal individual with the mouth open and in quiet breathing, the uvula generally points to the middle, and the palate and its arches have a uniform outline. In a hemiplegia, however, according to our investigations, in about 80 per cent. of the cases, the uvula generally points away from the paralyzed to the normal side, and the palatine arch, because of the relaxation or loss of tone of the muscle, loses its normal concavity and appears flatter and wider. In ten out of the hundred the arch appears to be narrower on the paralyzed side. But in these the weakness of the palate can be readily determined in phonation. Even in these cases, however, in comparison with the normal side, the palate always appears ironed out or flat.

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In the other ten patients we were unable to obtain a satisfactory view because of physical reasons.

When, however, the soft palate is put in motion by saying "ah," the weakness of the palate is at once apparent. Instead of the normal contraction, the response of the arch on the paralyzed side is slower, and what can be best described as the giving way or the relaxation of the palate is greater and in a direction backward and toward the normal side, as if the normal palate exerted its influence by drawing the back part of the paralyzed muscle toward it. There is, of course, a variation in the extent of the palate weakness; but some involvement is found in every case.

Tetzner,[6] in 1909, also studied the extent of the soft palate involvement in hemiplegics, and divided his cases into four groups. In a certain number he was unable to get a view of the palate. In the second group the soft palate was so completely paralyzed that it moved very little. In the third group the soft palate did not move in phonation, but did move symmetrically in gagging, while in the last group in phonation there was undoubted movement of the soft palate, as in fifty-two cases of cerebral hemiplegia he found that in forty, or 77 per cent., there was a drawing of the soft palate upward and *toward* the paralyzed side. In eleven of these cases the soft palate acted symmetrically.

These observations coincide very much with ours in the sense that the author found in most instances a weakness of the palate in phonation; but while he states that the movement of the palate is backward and toward the paralyzed side, our observation is that the movement is backward and toward the healthy side, quite the opposite conclusion. With the knowledge of Tetzner's observations, we have repeatedly proved to ourselves that our views are correct. Besides, in our study we observed the condition of the palate at rest, to which Tetzner paid no attention.

6. Tetzner: Neurol. Centralbl., 1909, p. 520.

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An interesting contribution to this subject, which establishes that our view is correct, is that by Horsley and Beevor,[7] who in their experiments found that with minimal electrical stimulation of the cortex of one hemisphere, elevation of the soft palate toward the opposite side could be obtained more frequently than bilateral elevation of the palate. It follows, then, that if stimulative experiment produces an elevation toward the opposite side, paralysis will cause a movement of the palate toward the sound side.

WEAKNESS OF THE TONGUE

The weakness of the tongue in hemiplegia is, of course, very well known. The generally accepted view is that in a right hemiplegia, for example, the tongue deviates to the right or the paralyzed side, because of the unaffected action of the normal muscle. The extent of this paralysis varies, and is generally most marked directly after a hemiplegia, and gradually disappears; but in some cases this weakness is always apparent. Beevor[8] investigated the tongue in hemiplegia. He was particularly concerned with the fact that the tongue moved to the paralyzed side, not only in a paralytic condition, but also as a result of a cortical irritation. He explained this phenomenon by the fact that the cortex revealed that there are two centers for the movements of the tongue, one in the upper part of the tongue area which caused deflection toward the opposite side, and another situated lower and more anteriorly, stimulation of which caused protrusion of the tongue in midline. The latter affects equally the movements on the two sides of the tongue, and it is the disturbance of this movement which causes the flexion of the tongue toward the hemiplegic side, and not the voluntary movement of turning the tongue toward that side. The latter is a unilateral movement, and Beevor has shown that it is affected in hemiplegia, the patient having more difficulty in putting the tongue

7. Horsley and Beevor: Phil. Tr. Roy. Soc., 1894, clxxxviii, 62.
8. Beevor: Brain, 1906, p. 487.

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into the cheek on the paralyzed side than in moving it in the opposite direction.

In our investigation we studied the position of the tongue when lying in its cavity, and its action when protruded. The extent of the weakness on the paralyzed side was generally commensurate with the extent of the palatine involvement. In almost every case the tongue was flabby to touch and pressure as compared with the normal side. When lying in its cavity, especially in its posterior part, it appeared higher and thicker, and its width was less. This was especially brought out when the tongue was protruded. In about 25 per cent. of the cases no change was found in the circumference of the tongue when at rest, while in a few, about 5 per cent., the tongue appeared smaller on the paralyzed side. When protruded, especially in early cases, the tongue moved to the side of the paralysis; but what was most apparent was the greater convexity or thickness and lessening of the width, especially in its posterior part.

Our investigations of the palate and tongue in diplegia were not producive, chiefly because of the lack of comparison with the normal.

In pseudobulbar palsy, our findings were practically those in hemiplegia; for in the majority of cases of pseudobulbar palsy, one side is always more involved than the other. In a few instances, especially in one case in which the bulbar symptoms were most marked, there was not the slightest doubt about the weakness of the soft palate. The tongue movements in pseudobulbar palsy are always limited, and the lack of tone noticed in hemiplegics is easily apparent.

CONCLUSIONS

1. In all hemiplegics in which there is an involvement of the lower part of the face, there is also weakness of the soft palate. This can be seen with the mouth open, with the palate at rest and in phonation. When at rest the arch of the palate is flatter and broader than that on the normal side. In phonation

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the soft palate on the paralyzed side appears to give way and the arch of the palate goes back and toward the normal side.

2. In hemiplegics in which the lower part of the face is involved, the tongue on the paralyzed side loses its elasticity, and is flabby to touch and pressure. When lying in its cavity, the posterior part especially appears to be thicker and not as wide in comparison to the normal side, this being especially brought out when the tongue is protruded.

1909 Chestnut Street.

ABSTRACT OF DISCUSSION

DR. FRANCIS X. DERCUM, Philadelphia: In an organic hemiplegia involving the leg, arm and face of one side, the tongue, when protruded, deviates toward the paralyzed side. In a hysterical hemiplegia it deviates toward the sound side. The physical character of the tongue as it rests on the floor of the mouth in organic hemiplegia, as well as the condition of the soft palate and half arches furnish additional points of value in the further differentiation between an organic and a functional hemiplegia.

DR. WALTER TIMME, New York: In phonation or in an attempt by the patient to move the soft palate, in which direction is his uvula carried—toward the paralyzed side or toward the sound side? It is my experience that the large majority, in contradistinction to Dr. Weisenburg's findings, move to the nonparalyzed side of phonation.

DR. GEORGE A. MOLEEN, Denver: One might say that in the examination of cases of hemiplegia the observation of palate and tongue is a routine. I have often, however, been struck by the point referred to by Dr. Weisenburg, that those cases which eventually prove to be not due to destructive lesions were associated with diminished or very slightly perceptible deviation of the tongue, and likewise the palate was but little influenced, while in those cases of organic hemiplegia it is ordinarily found that in phonation the palate is drawn to the opposite side.

DR. J. RAMSAY HUNT, New York: Were these observations made in old cases or in recent cases?

DR. THEODORE H. WEISENBURG, Philadelphia: We found that in hemiplegia the uvula points to the paralyzed side in phonation. Our cases were mostly old. In new cases the palate symptoms are much more marked than in older cases. So common has been the weakness of the palate that I would doubt the presence of hemiplegia without it.

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SOME CLINICAL STUDIES OF THE PROBLEMS OF CEREBRAL TONE

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During the last five or six years some important additions to our knowledge of muscle tonicity and the more generally inclusive topic of cerebral tone have been made by clinicians and pathologists. The clinical bearings of cerebral tone, which have usually been left for the discussion of psychologists, are becoming more and more recognized by neurologists, who are finding in their study some matters of practical value in diagnosis and treatment, especially the former.

In many forms of cerebral and spinal disease, as, for instance, in hemiplegias, diplegias, jacksonian spasms, perseveration, catalepsy, catatonia, atonic and spastic paraplegias, disseminated sclerosis, tabes, amyotonia, dystonia and Thomsen's disease, the presence and pathogenesis of muscle tonicity are of especial interest.

In a large majority of the cases of permanent hemiplegia — hemiplegia persisting as a marked loss of power for more than six months — the general picture as to contracture or spasticity is that well known to neurologists, and such as has been described in numerous treatises on nervous diseases.

Under the designations "early and late contractures," certain interesting phenomena of tonicity have been discussed by nearly all writers of neurologic textbooks and by many contributors to current neurologic literature. The so-called early contractures in hemiplegic cases are usually observed in apoplexy of marked severity, as, for instance, that in which a hemorrhage

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breaks through the cerebral substance into the lateral ventricles. These early contractures, as indicated by Oppenheim, are usually attributed to irritation of the pyramidal fibers. Developing within a few hours, if death does not supervene, as not infrequently happens, these may disappear after a short period, that is, in the course of a few days. The musculature of the paralyzed side of the body in these early contractures is affected generally, the spasticity not assuming the usual forms observed in the so-called late contractures.

Late contractures which have received much attention since almost the earliest days of the study of hemiplegia usually come on after a period ranging from two weeks to as many months. Sometimes they do not begin to be noticeable until three months after the apoplectic attack, but development as late as this is comparatively rare. The condition as to tonicity may be fairly regarded as permanent after the lapse of six months. It is generally held that permanent contracture increases with degeneration of the motor centers and tracts. While this is correct, it is now believed that it is not a sufficient explanation of such contractures, to say that they are due to secondary degeneration. In this connection it might be recalled that a number of competent observers have reported cases of hemiplegia and diplegia with necropsies and microscopic examinations, in which lesions of the pyramidal fibers were absent, although in some instances these fibers were of finer caliber than is normal.

Some revision of the descriptions usually given of cases of permanent contracture is desirable. The unusual differences as to spasticity and flaccidity (hypertonia and hypotonia) exhibited by different cases have received comparatively little attention. The excellent description given by Oppenheim[1] of the state of permanent contracture usually observed in hemi-

1. Oppenheim, H.: Text-Book of Nervous Diseases for Physicians and Students, translated by Alexander Bruce, ii, 687.

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plegia is correct so far as it goes, but there is no particular stress put on exceptional cases. Oppenheim says:

> The arm is usually in the following position: the upper arm is adducted, the forearm bent at a right or acute angle, the hand flexed and pronated, the fingers flexed either in all the joints or in the interphalangeal joints only. A contracture of the arm in the position of extension is rare. When the contracture is slight, the fingers can be extended. The leg is in the position of extension, the foot usually in the equinovarus position. Flexion contracture of the legs is very uncommon (cases of Prochátzka, David-Gallavardin, etc.), and occurs only under unusual conditions. The contracture seldom extends to the facial, and most rarely of all to the hypoglossus.

This description would answer in a general way for the cases of my series, but in a study of the question of spasticity and flaccidity it is not sufficiently explicit. It fails, for instance, to indicate that in some cases varying conditions of flaccidity and spasticity may be present in the same contractured limb.

A particular study of forty cases of hemiplegia was made. In the present preliminary contribution I shall not attempt to give any tabulated summary or elaborate consideration of the facts of tonicity exhibited by these patients, merely calling attention, for the purpose of awakening discussion, to some exceptional conditions.

In some cases of hemiplegia of several months' or years' standing, an admixture of flaccidity and spasticity will be found in the same limb, a fact which has some significance, especially in the diagnosis of the extensions of the lesion and the mechanism of the production of aberrant muscle tonicity. As a rule, if one part of the limb is spastic, as for instance when the musculature causes flexion at the elbow, the whole extremity will be set down in the notes as spastic or hypertonic, and yet closer examination will show in not a few cases that this is not correct. One or two examples may be given:

In a case of left hemiplegia of five years' standing in the intervals between voluntary efforts which could

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be made with the partially paralyzed upper extremity, the arm remained in a position of slight flexion at the elbow and was flaccid. The hands and fingers could be fully extended passively and could be moved in any direction. In them there seemed to be an admixture of hypertonicity and flaccidity, the former more so in the metatarsophalangeal joints. The case was evidently one both of loss of power (true motor paralysis) and irregular innervation. In this case the lower extremity was much more paralyzed and spastic than the upper, the hypertonicity being in proportion to the retention of power.

For many years I have been in the habit of noting the presence of flaccidity or extreme hypotonia in hemiplegic cases at different periods after the apoplectic attack. In most cases this hypotonia is marked at the time of the apoplectic onset and for a greater or less period (hours, days or weeks) afterward. It is not necessary for me to enumerate special observations, which would constitute a mere piling up of well known facts. One type of case not infrequently seen is illustrated by a patient recently observed:

This patient, a woman of middle age, suddenly became a left hemiplegic. Paralysis of the limbs was total and flaccid or flail-like. It continued without change as regards tonicity for a period of about twelve weeks, and then began to give evidences of oncoming spasticity, slight in degree, this gradually increasing and spreading, but never becoming marked, at least during an observation extending over several months. I have observed other cases, especially at the Philadelphia General Hospital, in which total flaccidity continued for several weeks or months and was then gradually replaced by spasticity or an admixture of spasticity and flaccidity.

Permanent flaccidity or total toneless paralysis, that is, absolutely atonic hemiplegia, is comparatively rare, and doubtless in different cases requires special explanation.

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A woman, aged 52, was examined seven months after the apoplectic attack. The left upper extremity was absolutely flaccid or atonic and powerless. The patient could not move the arm, forearm, hand or a single finger. When the limb was dangled, the hand assumed a wrist-drop position with the fingers somewhat flexed, this being due to the weight of the paralyzed hand. Tonic innervation was not present in this extremity. The arm as compared with the other arm was somewhat larger, this probably being due to lost vasomotor tone. The left lower extremity, like the left upper, was completely toneless and powerless. The leg lay rotated outward at the hip and was a helpless mass in any position in which it was placed. The whole lower extremity, like the upper, was somewhat swollen. No plantar reflex and no knee jerk could be obtained. Sensation appeared to be somewhat impaired, but not markedly.

The notes on the following case were made only three months after the oncoming of the paralysis, and of course the conditions as to flaccidity and hypertonicity may have changed later. However, the case is instructive as showing the continuance of complete flaccidity in the upper extremity for at least the time named, while a spastic condition was present in the lower limb. In both the upper and lower extremities the motor paralysis was absolute.

The patient, a woman, aged 47, was paralyzed on the left side of the body without unconsciousness. The left side of the face dropped. The left upper extremity was completely powerless and toneless. Sensation was retained in this extremity, and muscle and tendon jerks were plus. Wrist clonus could sometimes be elicited. The left lower extremity was kept somewhat extended and was rigid at the knee. It was not possible to throw the leg into different positions, as in the preceding case. Sensation was retained and a Babinski response was present. It was difficult to bend the leg at the knee, although this could be done by the exercise of considerable force.

Quite recently I observed a case of total flaccid left hemiplegia. The toneless paralysis was comparatively of recent origin, but so complete as to be worthy of special attention. A fuller report of the case will probably be made later.

The most important symptoms in this case were intense and frequently recurring pain in the head, nausea and projectile vomiting at times; papilledema in a comparatively early

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stage, and on the focal side, complete atonia or flaccid paralysis of the left upper and lower extremities with some paresis of the face of cerebral type, jacksonian epilepsy beginning in the left side of the face, astereognosis, impairment of cutaneous and muscular sensibility, and somewhat increased deep and superficial reflexes without, however, a Babinski response. The patient showed also considerable mental hebetude.

Necropsy in this case showed an abscess occupying the greater part of the parietal lobe. It extended to the motor region, but as far as Dr. William G. Spiller could determine at the time of the gross examination made by him, it did not extend into this region. In extent the abscess was from above downward about 2 inches, from before backward about the same, and from within outward about an inch and a half in diameter. The lower level of the abscess was just below the upper portion of the callosum, and it reached close to but did not involve the cortex.

This case is of particular interest in connection with the question of the part played by sensory impulses in the development of cerebral tone. Hypotonia or flaccidity is one of the commonplace observations in cases in which the sensory pathway from the periphery to the cortical areas concerned with sensation are largely destroyed. One needs but to recall the common condition present in ordinary advanced tabes and destructive lesions in various positions along the sensory pathway, as in the tegmentum, thalamus and parietal lobe which have been recorded in cases of flaccid palsies, these being in most cases associated with more or less anesthesia. The stimuli which reach the frontal lobe from the periphery and are the constant feeders of cerebral tone are by such lesions cut off, and tonic innervation suffers.

Now and then a case is encountered in which the tonic innervation is of such special form as at once to attract attention. In most cases after several years' standing the upper limb will be strongly contractured in flexion at the elbow with flexures also at the wrist and fingers. Occasionally, however, a case is seen in which the entire upper extremity is in a condition of hypertonicity, and this so generally expressed in the extensors and flexors alike that the limb continues in an extended position.

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For example, in a middle-aged hemiplegic whose paralysis was of about four years' standing, the arm was extended and hypertonic. The hand was contractured and somewhat flexed at the carpus and extended at the metacarpophalangeal joints.

Perseveration or "tonic innervation" is the subject to which neurologic attention has been directed largely through the observations and discussions of Liepmann, and Kinnier Wilson and Walshe.[2] The latter have defined this term "as the inability owing to a central lesion to relax innervation in any muscular group or groups." The term was invented by Liepmann, who first described it in connection with a case of apraxia.

Wilson and Walshe describe three personal cases:

In the first two "tonic innervation" or perseveration was present in the limbs of the left side, especially in the upper extremity. In the same cases evidence was present of involvement of the corticospinal pathway in the form of hemiparesis and slight spasticity. In both of these cases the symptoms were left sided. The general symptoms of brain tumor were present. In both cases operation was successfully performed, and endotheliomas of large size removed, the operations resulting in complete relief from the perseveration, but leaving marked postoperative hemiplegia. Apraxia was not present in either case.

In the third case, "tonic perseveration" and marked motor apraxia were exhibited associated with paresis and spasticity of moderate extent, indicating implication of the pyramidal system. The perseveration in this case was illustrated, especially in the inability of the patient to relax after grasping objects or closing the hand, but the tonectic phenomena were also well shown in movements of all parts of both the upper and lower extremities, although much more decidedly in the upper. The tumors revealed and removed by operation in the first two cases were so situated as to involve the superior and middle frontal convolutions and their subcortex from a point just in front of the central fissure forward. In the third case operation was not performed, and the exact nature of the lesion present was not determined.

Besides the case of Liepmann, Wilson and Walshe refer to other cases of Steinert, Van Vleuten, Kleist, Kurt, Goldstein, Kroll, Coriat, Rhein, Macfie Campbell, and Pick.

2. Wilson, S. A. Kinnier, and Walshe, F. M. R.: The Phenomenon of "Tonic Innervation" and Its Relation to Motor Apraxia, Brain, 1914, xxxvii, Part II.

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In true "tonic innervation" the volitional act is the disturbing element. The fundamental defect in "tonic innervation," according to Wilson and Walshe, is not of a psychic but of a psychomotor nature.

They emphasize the fact that "tonic innervation" and apraxia are separate and independent symptoms, although they may occur together.

An important fact in connection with the study of "tonic innervation" or perseveration is that which is indicated in the statement of Wilson and Walshe "that the corticospinal system for the limb in which 'tonic innervation' is found is not functionally normal: at the same time the involvement must not be too great. "As a corollary," these writers say, "the remark may be made that tonic innervation is not dependent on, nor does it proceed *pari passu* with, hypertonicity of pyramidal origin."

Wilson and Walshe make an interesting comparison and draw analogies between "tonic innervation" and myotonia congenita or Thomsen's disease and myotonia atrophica, holding that "as far as the actual symptom of inability to relax a contracted muscular group is concerned, no clear or unequivocal distinction can be drawn between its manifestation in the latter of these conditions as opposed to the former." My own observations which cover cases of each of these different forms of aberrant tonic innervation are confirmatory of this view. I am firmly of the opinion that the phenomena in Thomsen's disease and in myotonia atrophica are not myogenic, but of central (cerebral and spinal) origin.

In conclusion I shall give in detail the notes of a most instructive case of perseveration:

A. L., a white male, aged 37, unmarried, a marine, while located in the Panama Canal Zone, had an attack in July, 1912, of hemiplegia or hemiparesis accompanied by aphasia. The attack was not connected with any injury and came on while the man was at luncheon. He remained in the Ancon Hospital about six months, during which time he relapsed

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several times with fresh cerebral attacks, probably hemorrhagic in character. His hemiplegia almost entirely cleared up; the aphasia only in small part. Dr. W. E. Deeks, who conveyed these facts in a letter, also stated that he had a pyelonephritis due to a *Bacillus pyocyaneus* infection. He had at the time when he was first seen by me jacksonian epileptic seizures involving chiefly the right arm. When the examiner offered to shake hands with him he refused, intimating by pantomime that he was afraid he would injure the proffered hand. His reason for this was quickly seen when he was offered the handle of a percussion hammer. This he grasped tightly, and it could not be dislodged from his hand for some time. He was then told to make a fist, and when told to relax the hand he could not do so, and the examiner was unable with his two hands to unclasp the fingers.

Examination of the upper extremities showed that on the right side the triceps and biceps reflexes were increased, being normal on the left side. Plantar stimulation caused plantar flexion on both sides. On the right side plantar stimulation caused a reflex of defense, but not extension of the great toe. Pain, touch, heat and cold senses were normal. In walking, the right foot dragged slightly. Station was unaffected with the eyes open or closed. Mentality was good. He was a motor aphasic, having a few short expressions only.

An eye examination was made by Dr. H. M. Langdon, who reported that the pupils were equal, regular, and reacted to light and accommodation; $\frac{6}{10}$ normal vision on illiterate card; media clear; disks normal in color and outline; no changes in the fundi; no hemianopsia, and cranial nerves normal.

Wassermann examination proved negative, as did also the general investigation of the blood and examination of the urine.

Shortly after he was admitted to the nervous ward of the Hospital, Dr. William G. Spiller happened to be in the ward when this patient had an attack of jacksonian spasm, preceded and accompanied by phenomena of perseveration. He dictated at once the following account of the seizure:

"When first seen by me the hands were firmly clasped together, and it required much effort to separate the fingers even of the left hand. So soon as the hands were separated the left hand was found to be in a normal condition. There was no spasticity of this hand, and yet while the hands were clasped it required effort to raise the left fingers, from which it seemed that there was also spasticity in the left hand. It is remarkable, however, that immediately after the left hand was released there was no spasticity in this hand. The right hand remained in strong clonic spasm. In a few minutes the right fingers could be moved voluntarily very slowly and to a very limited degree. During the early part of the attack the right lower limb was not affected, but five or ten minutes after the beginning of the observation the right lower limb

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presented a tremor involving all parts of the limb, but not causing displacement of the limb at any joint. The right upper limb then presented clonic spasm involving the whole limb and lasting possibly two or three minutes. This then yielded to a tonic spasm. The right hand seemed to be more involved in the tonic spasm than any other part of the limb. The attack lasted fully twenty-five minutes, and at the end of this time there was fine tremor of the whole right upper limb and whole lower limb. At this time weak movements could be made voluntarily of the whole upper limb."

Although the general symptoms of brain tumor were absent, in the light of the cases of Kinnier Wilson it was thought that possibly a tumor or cyst might be present in the left midfrontal region, and it was believed that an operation could at least do no harm and might give some relief. Dr. Charles H. Frazier operated, Jan. 27, 1915, making a left frontal osteoplastic flap. No tumor or meningitis was discovered, but the brain presented a somewhat unhealthy appearance with lack rather than excess of pressure and slightly sunken outlines. It was probable that the patient had suffered from an attack of infectious encephalitis with repeated small hemorrhages. The symptoms presented after the operation were practically the same as those before, with the exception that there seemed to be less dragging of the right leg.

The following is a summary of notes made about three months after the operation:

The patient's station and gait were good. He was quite firm and steady and did not tend to drag either foot. Examining the right upper extremity and comparing it with the left, the patient had all movements of all parts of the limb—shoulder, arm, forearm, wrist, hand, etc. The left upper extremity, like the left leg and left side of the body generally, was practically normal as to power, testing him for strength by duplicated active movements. Astereognosis was not present.

He could say a few things such as "No maam," "Can do it," "Yes," "No," "This here," etc.

When tested for apraxia with a key, dumbbells, opening a knife, and sharpening a pencil, etc., he performed all the tests well with the exception of the dumbbells. In this test he was supposed to place the dumbbell held in his right hand on his left shoulder. In attempting to perform this he either placed the dumbbell behind his head or on the left side of his neck.

When the patient flexed the right forearm on the arm, not only the biceps went into powerful contraction, but also the triceps. During the examination the patient's right hand suddenly became flexed, and the muscles of the forearm, both flexors and extensors, became hard. This persisted for two minutes by actual timing, and during the attack he was

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unable to relax the innervation. At the end of two minutes the fingers slowly extended, and the muscles of the forearm relaxed. The muscles of the left arm and the lower extremities were not involved; neither was the face, and there was no disturbance of consciousness.

Directed to flex his forearm on his arm, the patient began and continued to do this with the appearance of great effort, the arm slowly flexing against resistance and with the apparent help of other muscular groups than those usually employed. On examination of the extensor muscles (triceps, etc.), it was found that these were in active tension and contraction, showing that the difficulty in flexion appeared to be due, in part at least, to the fact that the excessive tonic innervation was universal, or was both of the extensors and flexors of the forearm. In other words, the extensors were not in the relaxed condition usually found when strong flexion of the arm occurs.

Many years ago (1886) I recorded a case of myotonia which at that time I regarded as of hysterical origin, partly because the patient was cured or vastly improved as the result of treatment, this being the repeated applications of the actual cautery to the back of the neck.[3]

In 1891 I[4] published an account of two remarkable cases illustrating tonic innervation and tonectic phenomena of unusual interest.

One of these was probably an instance of Thomsen's disease, although I could not obtain any family history. All parts of the musculature of the body were liable to sudden "locking." The patient had many falls as the result of the sudden access of what might be termed universal active perseveration. In opening and shutting the hands, in opening the mouth, in rising from a recumbent or sitting position, and in numerous other ways, all of which are detailed, some of them with photographs, this patient presented in extreme degree the phenomena of tonic innervation usually induced by volitional effort, although coming on at times without apparent interference of the will.

In the same article in which this case is recorded I published an account of another case of myotonia and athetoid spasm which is cited by Wilson and Walshe, who speak of it as difficult to classify, while

3. Mills, C. K.: A System of Practical Medicine by American Authors, edited by Drs. William Pepper and Louis Starr, v, 241.
4. Mills, C. K.: Myotonia and Athetoid Spasm, International Clinics, April, 1891, Series 4.

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regarding it as unique. These writers say that the indications all point to the lesion being of central origin. In their search for literature they overlooked the fact that subsequently an account of the necropsy on this patient was published by me.[5] Besides the myotonic and athetoid phenomena this patient had marked disturbances of sensation. The following partial summary of the findings at the necropsy shows that both an irritative and destructive meningoencephalic lesion extended over a large part of the convexity of both hemispheres, the pathologic conditions being most pronounced in the parietal region:

The calvarium was dense and heavy. The dura was unusually adherent and nowhere translucent. The pacchionian depressions were not unusual. Over the central part of the lateral surfaces of both hemispheres, that is, chiefly over the superior and inferior parietal gyres, the dura was adherent to the pia. The pia was deeply injected, thickened, and infiltrated with plastic lymph, and in numerous places were more or less firm attachments to the brain substance; but the injection, infiltration, exudation and adhesions were much more marked in the posteroparietal regions than elsewhere.

Beneath the area of meningitis, both the cortex and the subcortex were softened, giving the appearance of a sunken subcortical or subpial cyst. On the opposite (left) side of the brain in a nearly corresponding but somewhat smaller area was a similar belt of inflammation and softening. Subsequent incisions on both sides showed that the softening included the whole of the gray matter, and involved to a considerable extent the white, but did not invade the ganglia or capsules; it was confined to the supraventricular corona radiata.

The vessels at the base were somewhat stiffened, but no other brain lesions were discovered.

As indicated in the previous notes of a series of cases of hemiplegia observed, variations in the degree and character of the permanent contractures, or the entire absence of contractures, were matters of paramount interest. Numerous theories as to the occurrence of persistent contractures in hemiplegia have been advanced and discussed, but none of these have proved entirely satisfactory. Some of the older observers

5. Mills, C. K.: Jour. Nerv. and Ment. Dis., December, 1891.

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simply contented themselves with the statement that the contractures were due to the secondary degeneration following lesions of the corticospinal motor system, or that they were dependent on irritation of the neurons of this system remaining by the products of the degenerated fibers. In most of the theories advanced, the incompleteness of the destruction of the pyramidal apparatus has been given as a partial explanation of the contractures. With complete destruction of the pyramidal tracts, as in total transverse paraplegia and rare cases of total hemiplegia, flaccidity or atonia are the striking manifestations, as was illustrated in some of the cases studied for this paper. It has been held, in other words, that the contracture was in a degree proportionate to the amount of paralysis present.

In the consideration of such questions as contracture and aberrations of muscle tonicity in general, the result of organic lesions (hypertonia, hypotonia, dystonia, perseveration) attention is too strictly confined to the pyramidal motor system. It is true, of course, that through the corticospinal motor apparatus and its musculature, the phenomena of muscle tonicity are exhibited, but it does not follow that tonus is primarily a pyramidal quality. The whole subject of aberrant muscle tonicity is much simplified by the recognition of the existence of a tonectic apparatus, that is, an apparatus for maintaining tonic innervation, extrapyramidal and therefore structurally distinct from the pyramidal system, although necessarily connected with it. In a previous paper[6] I have endeavored to present this view.

Tone is necessary for the rhythmetization of movement both voluntary and automatic. Tone, at least in the human being, is not a spinal or cerebellar attribute, but belongs to the cerebral cortex, becoming more cortical, as Crocq has shown, as we ascend from animals lower in the scale, the frog, the dog and the ape, for instance, to man. That the

6. Mills, C. K.: Muscle Tonicity, Emotional Expression, and the Cerebral Tonectic Apparatus, Neurolog. Centralbl., 1914, No. 24.

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cortex of the cerebrum is the seat of tone is not a new idea. It has been held by many, the views of those expressing the theory, however, being somewhat diverse. In most instances the pyramidal motor cortex seems to be regarded as the rendezvous and storehouse of the tone derived from sensation and idea. The cerebral tonectic apparatus, I believe, is structurally separate from the motor projection systems. It has special cortical areas of representation, and these are connected by fiber systems with the sensorimotor executive apparatus on which tonectic excitations exert their influence. Regarding the frontal cortex from the central fissure to the anterior pole of the brain as subdivided into a prefrontal, a midfrontal and a postfrontal (motor) region, the cortical tonectic zone is chiefly midfrontal. The striatum is an association or integration region where are regrouped the excitations from the differentiated cortical centers for tone. Wilson holds that higher (cerebral) centers concerned with tone must be conceded, and that there is increasing evidence that muscular tone depends to some extent on the reaction between the "basal ganglia" and the cortex and especially the intermedioprecentral cortex.

The midfrontal or intermedioprecentral region is the cortical seat or rendezvous of tone, the place to which stream the affects or tone impulses of the sensory centers of the cortex. Separate cortical tonectic centers constitute together a cortical tonectic zone. This area is not necessarily topographically entirely segregated from the pyramidal motor cortex. Each motor center probably has its separate outlying cellular tonectic annex or dependency, these centers of tone lying in the main anterior to the motor centers proper.

I need not further recall the data and views expressed in this paper on muscle tonicity. Admitting its validity and turning to the question of persistent contracture or any other phenomenon of aberrant muscle tonicity, as perseveration, the explanation of the conditions presented becomes comparatively simple. In the first place, if tonic innervation is supplied to the pyramidal system and exhibits itself through the activities of this system, it is clear that for normal rhythmetization of movement the pyramidal system itself must be intact. Pyramidal lesions, according to their location and extent, will interfere with normal tonic innervation. A complete transverse lesion of the spinal cord or of the internal capsule will block the transmission of tone-maintaining impulses to musculature innervated below the seat of the lesion. This would

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seem to be the simple explanation of the cases of flaccid or toneless paraplegia from transverse spinal lesions, and of at least some of the cases of flaccid hemiplegia from completely destructive capsular lesions.

When, as most frequently is the case, the lesions of the cord or of the internal capsule are incomplete, the tonectic apparatus remaining intact, innervation will be irregular or aberrant in its manifestations. Like the motor cortex, the pyramidal tract itself has its functional subdivisions, and according to the retention or loss of the fibers of these subdivisions, will be the residual spasticity or flaccidity (hypertonia and hypotonia). Herein lies the explanation of many of the irregular phenomena of muscle tonicity exhibited in permanent contracture.

The hemiplegic musculature may be variously affected in other ways than simply by lesions of the pyramidal tracts. The withdrawal from the sensory pathway of the peripheral stimuli which contribute to cerebral tone by destructive lesions situated anywhere in the sensory pathway will eventually influence muscle tonicity. This is exhibited in the phenomena of hypotonia presented by advanced cases of tabes, or again as in one of the cases cited in this paper by a large destructive lesion of the parietal lobe.

Lesions of the tonectic cortex (chiefly the midfrontal cortex) or of the striatum, will give aberrant tonectic manifestations. The phenomenon of perseveration is particularly interesting in this connection. The cases so far recorded with necropsy support would seem to show that for its exhibition extrapyramidal lesions are usually to be sought for — lesions of the midfrontal (intermedioprecentral) cortex, of the striatum, or possibly of the ruber or thalamus. Usually in well marked examples of perseveration such as are referred to in this paper and have been recorded by Wilson and others, some involvement of the pyramidal system is indicated, although this is generally of moderate

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extent, the lesions being chiefly extrapyramidal. It was noteworthy in the series of cases studied for this paper that perseveration was rarely present, which is probably best explained by the fact that, in most of the cases of the series, destruction of the pyramidal tracts, while not complete, was large in extent. It is probable that in a few of the cases in which the contracture was exhibited, not in the usual irregularities of spasticity and flaccidity, but in a generalized excessive tonic innervation, the lesion present was in part at least extrapyramidal as well as pyramidal.

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ABSTRACT OF DISCUSSION

DR. WILLIAM J. HICKSON, Chicago: In making a diagnosis of hemiplegias and diplegias in the feeble-minded, I used the blood pressure apparatus in over 100 cases and invariably found marked changes in the registration even where it was very hard to make a clear cut diagnosis of spastic or flaccid paralysis. In regard to flaccid paralysis, for over a year I had opportunity to work with a well known authority and saw the outcome of his cases on which he was at that time specializing, and after all the work and manual training carried out in handling that sort of thing these cases were invariably worse, at the end of a year or two they had this tonicity which Dr. Mills has so well illustrated.

DR. JULIUS GRINKER, Chicago: I have in mind the various cerebral palsies of long standing, that show, in old cases, a certain degree of flaccidity combined, perhaps, with the movements that are called athetoid. In many of those cases the lesion was found either in the cortex or the subcortex. At the present time I have under my care a case similar to the one that Dr. Mills used as a basis for his hypothesis. A young man of about 19 came to me complaining of the symptoms of paroxysmal hypertonic contraction of the toes, preventing him from walking, for when he attempts to walk there occurs a sudden dorsal flexion of his big toe first in one foot and then in the other. He, too, presented the symptom of perseveration, or persistent muscular contraction in the muscles of the lower extremities. In recommending operative treatment in this case, I had in mind the probability of a cerebral lesion of obscure origin as to onset. It occurred to me that perhaps the operation of cutting certain posterior roots in the lumbo-sacral cord might benefit the patient. Dr. Elsberg of New York operated on the boy about four months

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ago and the case is much improved. While the question of therapeutics is not relevant to this discussion, yet the fact that the patient is improved would tend to confirm my diagnosis of cortical or subcortical lesion. I could not believe that I had a lenticular lesion in that case. Dr. Mills has thrown much light on my case, and I believe there are many other cases of disordered motility which his theory will explain.

Dr. Foster Kennedy, New York: I saw Dr. Grinker's patient. I, too, advised operation, but I did not believe the patient had had an encephalitis. So far as I could ascertain, the onset of the extraordinary rhythmic involuntary contractions of his leg muscles developed very slowly and progressively over a number of years, and I did not feel that they could be due to any encephalitic process; but on the contrary I believed there was a distinct identification with those cases which Dr. Hunt speaks of, as progressive torsion spasm of the pelvic muscles. Cases of obscure muscular tonicity of such character, without apparently any definite cerebral symptoms, occur quite gradually and are progressive in type. I am very much interested in the fact that operation was beneficial in that case.

Dr. J. Ramsay Hunt, New York: There can be no question but that the corpus striatum is a lesser motor mechanism which has to do with the control of associated and organic movements. And one of the essentials of that control is the control of tonus, because, as we have seen in Wilson's writings, if it is destroyed there is not only a paralysis, but a loss of control of tonus. With destruction of the cortex or pyramidal system, which is a much greater system, there is a loss of tonus. From our present knowledge therefore we cannot dissociate the loss of tonus from the loss of control. Dr. Kennedy has referred to my subject of progressive torsion spasm. It would seem that this is a disease of a lower tonus mechanism, even lower than the corpus striatum. This is evident when we stop to consider that as the nervous system has gradually developed in the course of evolution, there must and should be such a center as would serve to control tonus in automatic and associated movements. And it seems to me that there is good reason for believing that this mechanism is undergoing degeneration in such a disease as progressive torsion spasm.

Dr. Charles K. Mills, Philadelphia: The whole subject of tonus has been pretty thoroughly studied in the lower animals and up to man, as for instance by observers such as the Belgian, Crocq. My own views I tried to express—perhaps not so clearly—in the introductory remarks to my paper. About one thing I desire to be quite definite. I do not believe that in strictness tonus is a fundamental quality of the pyramidal system. If we are discussing a question like muscle

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tonicity, however, it is of course perfectly evident that muscle tone so considered can only be exhibited through a neuro-muscular apparatus of whch the pyramidal cortex and the pyramidal tract are a part. There is a tonetic system, both cortical and ganglionic, distinct from the pyramidal apparatus. The tone, however, must, so to speak, be delivered to the pyramidal system. In the cortex are differentiated tonetic centers and the striatum, or a part of it, is a structure, one of whose functions is to associate cerebral tone so that it may be applied to synergized movements. In man the centers concerned with tonic innervation are primarily cerebral and cortical. These centers of tone are charged by the inflow of sensation from the periphery, and also by the influx of ideational impulses, these being themselves primarily derived from sensation. As to the question of pelvic dystonia, as far as the very few cases I have seen are concerned, I do not believe the phenomena can be explained on any purely pyramidal theory, although it is always to be remembered that tonectic phenomena are expressed through the pyramidal system and its neuro-muscular connections.

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CEREBELLAR LOCALIZATION

AN EXPERIMENTAL STUDY BY A NEW METHOD *

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Unlike the doctrine of cerebral localization, which was founded on direct experimental evidence, the motor responses being obtained on stimulation of the cerebrum, the theory of cerebellar localization was evolved through studies in embryology and comparative anatomy. It was first formulated, in 1903, by the Dutch anatomist Bolk[1] who, from a study of the cerebellum in numerous types of mammals, deduced the theory that the developmental variations of its different lobules bear a relationship to the various muscle complexes in the body. Bolk, for example, noted that the lobulus simplex (his hypothetic center for the musculature of the neck) is especially well developed in the giraffe, and the lobus anterior (his center for the musculature of the head) in the elephant with his large proboscis; whereas the formatio vermicularis (the center for the musculature of the tail, according to his theory), is in the latter animal, as well as in the anthropoid apes and man, very small and rudimentary, but, on the other hand, is large and markedly lamellated in the cetaceans (sea mammals, as the dolphine, etc., which, like all fishes, possess long tails).

Bolk noted further that the developmental variations of the lobules depend not so much on the size of the muscles as on their functional capacity. Starting with the hypothesis that the cerebellum represents not

* From the Hull Physiological Laboratories, University of Chicago.
1. Bolk, L.: Das Zerebellum der Säugetiere, Jena, 1906. For other articles by Bolk, dealing with this subject see Footnote 2.

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muscles, but movements, in other words, the effects produced by a number of muscles acting collectively and harmoniously, he divided the organ into two types of centers, one, which he localized in the unpaired lobules of the vermian portion for the coordination of muscle groups in their bilateral activities; the other, which he localized in the paired lobules of the hemispheres for their coordination in unilateral and individualized activities. In this manner he explained the extraordinary size and foliation of the crus primum (the lobuli semilunares superior et inferior and the lobulus gracilis) in man, it being due, according to him, to man's great capacity for individualized and delicate movements with the upper extremities. Thus he also explained the ordinary size in man of the lobuli a and b of the lobus medianus posticus (the nodulus and uvula), the center for the musculature of the perineum, and the comparatively small size of the lobus anterior (the lingula, lobulus centralis and culmen monticuli with their lateral extensions), the center for the musculature of the head (according to his theory), these muscles acting bilaterally in practically the same degree in men as in the lower animals.

It is obvious that the theory requires for its verification experimental proof. The theory also presupposes that the function of the cerebellum is to coordinate muscle groups in their synergistic action. This is a supposition which is still subject to dispute, a point raised by Van Rynberk.[2]

A good deal of experimental evidence has, indeed, been since adduced in favor of cerebellar localization. The evidence is, however, far from conclusive, especially as regards the exact demarcation of the different centers, the results obtained by various investigators having been inconstant, frequently ambiguous, and, in some cases, directly at variance with one another.

2. Van Rynberk, G.: Ergebnisse der Physiologie, 1908, vii, 675, Asher-Spirós, Wiesbaden; ibid, 1912, xii, 536. Appended to these articles will be found an extensive bibliography bearing on the question of cerebellar localization.

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PREVIOUS METHODS OF STUDYING CEREBELLAR LOCALIZATION

Stimulation Experiments.—The striking success that has attended the electric method of excitation in mapping out the various motor zones of the cerebrum has naturally led investigators to utilize the same method for the study of localization in the cerebellum. The first to study the problem by this method was Ferrier,[3] who tested the electric excitability of this organ in birds, fishes and mammals, including apes. His results were not, however, such as to warrant the assumption of a functional differentiation of the cerebellum, such as is known to exist in the cerebrum. The most constant phenomena he observed were ocular movements which varied in direction according to the part of the cerebellum stimulated. The fact that the ocular nerves and roots are in such close proximity to the cerebellum mitigates, however, against their value as localizing phenomena, the effects having been due doubtless to the spread of the current to these structures. This theory was maintained also by Lourié[4] and Rothmann,[5] both champions of cerebellar localization. Ferrier states that the movements of the limbs, which he obtained on stimulation of the lateral lobes, were sudden, spasmodic and of an indefinite character. Associated with these limb movements he observed a contraction of homolateral pupil, undoubtedly the effects of the diffusion of the current to the oculomotor nerves.

Among others who studied the problem in this manner were Wersiloff,[6] Pruss,[7] Negro and Rosaendo,[8] Clarke and Horsley,[8] Rothmann[5] and Uffenorde.[9] Their results, on the whole, were far from uniform

3. Ferrier, D.: Functions of the Brain, Ed. 2, 1886, p. 186.
4. Lourié, A.: Neurol. Centralbl. 1908, xxvii, 102.
5. Rothmann, M.: Neurol. Centralbl., 1910, xxix, 1086, 389, 1205; ibid., 1911, xxx, 168 (with Katzenstein), p. 1146; ibid., 1914, xxxiii, 1011; Deutsch. Ztschr. f. Nervenh., xli, 105; Berl. klin. Wchnschr., 1913, l, 336.
6. Wersiloff, N. M.: Neurol. Centralbl., xviii, 328.
7. Pruss: Abstr., Neurol. Centralbl., 1903, p. 268.
8. Clarke, R. H., and Horsley, Sir V.: Brain, London, 1908, xxxi, p. 121.
9. Uffenorde: München. Med. Wchnschr., 1912, lix, 1279.

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and frequently contradictory. For example, Wersiloff obtained movements of the hindlimb on excitation of the vermis superior, Pruss on excitation of the lobus quadrantus, and Uffenorde, of the alae lobuli centralis, while Rothmann evoked such movements from the posterior part of the hemispheres and vermis exclusively, and then only in association with movements of the forelimbs.

While these authors maintained that the cerebellum is electrically excitable, Clarke and Horsley,[8] employing a perfected technic, concluded that the cortex cerebelli is not excitable at all and that the effects obtained are the result of either direct or indirect stimulation of the intrinsic or paracerebellar nuclei, structures which, according to them, represent the motor organ of the cerebellum.

It is thus evident that the method of direct excitation, whether by mechanical, electric or chemical[10] means, has proved inadequate and unsatisfactory for the study of cerebellar localization, and in the words of Van Rynberk, it neither proves nor disproves the theory.

Extirpation Experiments. — Another method employed for the study of cerebellar localization, and one which has indeed furnished a number of valuable data in connection with this problem, is the method of partial exclusion, or the production of small, circumscribed lesions of the various lobules of the cerebellum followed by a study of such motor disturbances as resulted from them. This method, which has been employed by Luciani,[11] Van Rynberk,[2] Luna,[2] Lourié,[12] Hulshoff Pol,[2] André-Thomas and Durupt,[13] Rothmann[5] and others, has proved of great value, not

10. Excitation of the cerebellum by local application of strychnin has been studied by Magnini. In contradistinction to such excitation of the cerebrum this gave rise to no effect as long as the irritant was confined to the cerebellar cortex, and did not come in contact with the medulla. Magnini was therefore opposed to the theory of cerebellar localization. (Quoted by Van Rynberk, Footnote 2.)

11. Luciani, L.: Human Physiology, iii; Engl. Transl. by Frances A. Welby, New York, The Macmillan Company, 1915.

12. Lourié, A.: Neurol. Centralbl., 1907, xxvi, 652.

13. André-Thomas and Durupt: Neurol. Centralbl., 1914, xxxiii, 208 (ref.).

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only for the study of cerebellar localization, but also in extending our knowledge of the phenomena of cerebellar lesions. It disclosed a number of variations from the normal which had not been appreciated before, such as head astasia, the cock's gait and the "military salute," the animal when excited raising its forelimb to the level of the ear, first described by Van Rynberk. In general, these studies tended to confirm the theory of cerebellar localization as postulated by Bolk. They differed, however, as to the strict localization of the various centers. The reason for this is to be found in the fact that the disturbances resulting from very small and circumscribed lesions of the cerebellum are exceedingly slight, if detectable at all. In my experiments on the cat, after the production of small, circumscribed lesions in the lobules of the hemispheres such as are reported in this communication, no disturbances whatever were observed, either in station, gait or general motility of the animal. The animals, in my experiments, aside from their enfeebled condition as a result of the operation, which in the case of the cerebellum is always one of great severity, showed no deviation from the normal. They walked up and down an inclined plane, jumped from a height and stood up on their hind legs when food was held up in front of them in a perfectly normal manner. As is well known, Luciani, who had an extraordinarily rich experience with cerebellar extirpations, was opposed to the view of cerebellar localization. He maintained that the cerebellum is a homogeneous structure, all segments of which function alike, so that deficiency of one is quickly compensated by the remaining intact portions. This is in entire accord with my observations, so far as they relate the gross phenomena generally looked for.

From these facts it is clear that neither the stimulation experiments nor the extirpation experiments have furnished the undoubted proof of a functional differentiation in the cerebellum, that they furnished for

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establishing localization in the cerebrum. This, I believe, should point to a fundamental difference in the function of these two organs.

THE AUTHOR'S CONCEPTION OF THE CEREBELLAR FUNCTION

In a recently published communication[14] I advanced the view, based on experimental evidence which I obtained by galvanometrically determining the electric potential on the two sides of the body after unilateral oblation of the cerebellum, that the cerebellum does not, as is generally assumed, act motorially on the periphery, but that it acts primarily on the motor and tonus centers of the encephalon (motor cortex of the cerebrum, paracerebellar nuclei and possibly also the nuclei of the midbrain), its function being to inhibit or control and regulate the activity of these nuclei, and that the phenomena of cerebellar deficiency are therefore to be interpreted as phenomena of hyperactivity of the latter structures. The cerebellum, according to this view, is a purely afferent mechanism, bearing in a broad sense the same relationship to the motor (and tonus) nuclei of the encephalon that a posterior root ganglion does to the motor cells of the anterior horn of the cord.

If this conception of the cerebellar function is correct, our failure to obtain constant and definite responses on excitation of the organ at once becomes clear. The cerebellum being an afferent, sensory structure, the motor effects obtained on its excitation are obviously due to the transfer of the stimulus to the subjacent structures, and not to stimulation of the cerebellum itself, and are therefore vague and indefinite, just as the diffuse and ambiguous movements obtained on excitation of the central end of a divided posterior root are due primarily to the effects of the stimulus on the motor cells of the cord. The fact (and this is admitted by all observers) that a far more

14. Meyers, I. L.: Galvanometric Studies on the Cerebellar Function, THE JOURNAL A. M. A., Oct. 16, 1915, p. 1348.

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powerful current is required to evoke a motor response from the cerebellum than is required for a similar response from the cerebrum also lends support to this view. It is well known that with powerful currents motor responses are also obtained from the sensory regions of the cerebrum, such as the parietal and temporosphenoidal lobes, the effects being admittedly due to the diffusion of the current to the neighboring motor zone.

To follow up this argument, if the conception of the cerebellar function as presented in my last communication is correct, the question of cerebellar localization should be studied through the medium of the motor cortex of the cerebrum, paracerebellar nuclei, etc., in order to determine whether or not after a circumscribed lesion of the cerebellum, the muscle group which, according to Bolk's hypothesis, is related to the site of the lesion, is in a state of functional hyperactivity, and also, if such hyperactivity is present, whether or not this is absent in other muscle groups.

To take a concrete example: The functional relationship of the musculature of the leg with the leg area of the contralateral hemisphere of the cerebrum is determined, after a lesion of the latter, by the spasticity (increased reflexes, muscular rigidity and clonus) of the leg affected; in other words, by phenomena of hyperactivity of the motor cells in the lumbar enlargement, due to their release from inhibition by the mutilated leg center in the cerebrum. Similarly, the functional relationship between the musculature of the leg and the homolateral crus secundum (Bolk's hypothetic leg center in the cerebellum) should be found in phenomena of hyperactivity of the leg center, and no other, of the contralateral hemisphere of the cerebrum caused by its release from inhibition by the mutilated crus secundum. Clinically, such hyperactivity of the cerebrum exhibits itself in disturbances of ataxia, the cerebellar gait, disturbances of equilibrium, hypermetria, etc.

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The symptoms described do not appear in any but severe lesions of the cerebellum and are not therefore available for the study of cerebellar localization. The state of activity of the various motor centers of the cerebrum, and also that of the tonus centers in the medulla[15] (the paracerebellar nuclei, which Thiele[16] by his studies of decerebrate rigidity demonstrated to be the tonus centers for the musculatures of the body) might be studied, however, by another method, namely, by noting the effects of exciting these centers by oil of absinthe, a convulsant which is known to exert its influence on these particular centers. The poison, owing to this particular mode of action, which has been established by the researches of Magnan,[17] Bechterew,[18] Gotch and Horsley,[19] Rubert Boyce,[20] Leonard Hill,[21] Bouché[22] and others, has been extensively employed in studies of brain physiology. When injected into the circulation, this poison produces convulsions which affect all the muscles of the body, are accompanied by unconsciousness, are of both clonic and tonic character, and present an appearance strikingly similar to that of convulsions in idiopathic epilepsy. These studies demonstrated that the clonic convulsions, which, on direct excitation of the cerebrum, are obtained only from the motor cortex and not from the corona radiata or any of the subjacent structures, are due to the effect of the poison on that part of the brain. Excision of the motor cortex of one hemisphere eliminates or reduces to a minimum the clonic convulsions on the opposite side of the body. Leonard Hill beautifully illustrated this fact by varying at will the clonic or tonic character of the con-

15. The state of activity of the various motor centers exclusively could probably be determined also by direct excitation of the cerebrum, or the galvanometric determinations of their electrical potential.
16. Thiele, F. H.: Jour. Physiol., 1905, xxxii, 355.
17. Magnan, V.: Arch. de physiol. norm. et path., 1873, v, 115.
18. Bechterew, V.: Neurol. Centralbl. 1895, xiv, 394.
19. Gotch, F., and Horsley, V.: Phil. Tr. Roy. Soc. London, 1891, clxxxii, 511.
20. Boyce, Rubert: Brit. Med. Jour., 1893, ii, 1097; Neurol. Centralbl., 1894, xiii, 466; Phil. Tr. Roy. Soc., London, clxxxvi, 363-381.
21. Hill, Leonard: Phil. Tr. Roy. Soc., London, 1900, cxciii, 102-110.
22. Bouché and Horsley: Brain, London, 1906, xxix, 453.

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vulsions by alternately leaving open or clamping the arterial supply of the cerebrum. The tonic convulsions, on the other hand, are due to the effect of the poison on the tonus centers in the medulla. Section of the cord below the bulb promptly abolishes all convulsions, tonic as well as clonic, in all the muscles connected with it below the line of section (Boyce and others). The poison, introduced into the blood, is carried of course to all parts of the brain and acts simultaneously on all the centers that are influenced by it. The hyperactivity of a certain center should consequently be followed by a greater response in its corresponding muscle groups.

I accordingly carried out my experiments as follows: I first produced small, circumscribed lesions of certain lobules of the cerebellum, using cats in all the experiments. The organ was reached by cutting the skin over the occipital region in the median line, severing the large mass of muscles on the posterior side of the neck at their attachment at the lambdoid suture. and, after retracting them downward and outward as far as possible, opening the skull by means of a small trephine one fourth inch in diameter. I found that by this route only could I reach the lobulus paramedianus and the crus secundum without injuring at the same time the crus primum. An injury to the latter could, however, be produced by an opening just above the lambdoid suture. To produce a circumscribed lesion of the crus secundum I trephined the skull just above the foramen magnum, a few centimeters away from the median line. Caution should be taken in trephining here not to injure the medulla which generally produces instantaneous death of the animal. The opening exposes a small part of both the lobulus paramedianus and the lobulus circumcludens or paraflocculus, and a lesion of the crus secundum can be made by injuring the area just above the latter lobule and external to the former. An injury to the lobulus paramedianus can generally be made without much

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difficulty after trephining the skull just above the area described for a lesion of the crus secundum, and an injury to the crus primum, after making an opening directly below the lambdoid suture. To make a trephine opening, at a considerable distance from the median line, which would expose the crus secundum or the outer part of the crus primum, is not, as a rule, a safe procedure, as it is accompanied by a hemorrhage too severe for the animal to survive. Therefore, only the lobulus paramedianus and the inner part of the crus primum can usually be brought to view.

After I produced the desired lesions, I kept the animals alive for a period of time varying from a week to twelve days to make certain they would recover and that they were not suffering from meningitis which would give rise to generalized phenomena and render the results valueless. During this time careful search was made for any manifestations of cerebellar deficiency, but, as stated previously, whenever the lesions were small and circumscribed, there were no symptoms whatever which might have indicated the site of the lesion. Only the general phenomena resulting from the operation, hemorrhage and shock from loss of cerebellar substance were noted. When these phenomena subsided and the animals showed unmistakable signs of recovery, they were subjected to the action of oil of absinthe as follows:

The animal was placed under ordinary surgical anesthesia by means of a tracheal cannula.[23] It was then placed on a board, the ventral surface down and the limbs tied to hooks at each side so that limbs as well as body were extended to their utmost capacity. The trunk of the animal was tied securely to the board by means of two strips of bandage, which encircled it, one at the upper part of the chest, the other at its posterior end. This arrangement effectually prevents the

23. Profound narcosis generally prevents the appearance of convulsions, either because of the profound depression of the cerebrum or as a result of the low vitality of the animal. The latter view is probably correct.

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movements of the trunk during a generalized convulsion from being communicated to the limbs. It also prevents all possible voluntary movements. In a number of experiments added caution was taken by nailing the limbs to the board. This is unnecessary, however, if the limbs are thoroughly extended.

With the animal in this position, two symmetrical, representative muscles from the forelimbs, such as the extensores communes digitorum, and a similar pair of muscles from the hindlimb, the gastrocnemii, were cut at the points of insertion. These muscles had been separated, by means of the fingers, from the surrounding tissues as close to their origin as possible earlier in the experiments, and kept from drying and cooling by normal salt solution at the body temperature. Their lower ends were attached to silk threads, any one of which was made to pass over two pulleys, fastened to two ringstands as shown in Figure 1. The thread was then connected to muscle levers. The levers, which were weighted down by small weights of equal size (7 or 10 gm.) to keep the muscles under a fair degree of tension, and equally calibrated, were made to write on a revolving drum covered with smoked paper. In order to eliminate possible frictional difficulty, caution was always taken that the threads, except for contact with the pulleys, should be free throughout their course from contact with anything else in the apparatus. Similarly, in applying the salt solution to the muscles, care was taken that it be applied very gently in order not to disturb their tension. The oil of absinthe was then injected into the external jugular or popliteal vein, a drop at a time, and the convulsions, whenever present, were noted both visually and by their records on the drum. They were found to correspond. Before proceeding, however, with a discussion of the results in animals with cerebellar lesions, it will be necessary to study a myogram of an absinthe convulsion in a normal animal.

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A study of this myogram (Fig. 2) reveals the following characteristics:

1. The effect of the poison exhibits itself simultaneously in all the muscles of the body. All the muscles are in convulsions whenever the intensity of excitation is sufficient to bring about this motor effect, and are quiescent whenever the motor excitation is not sufficiently intense. This is a rule to which, in my experience, there are no noteworthy exceptions.

2. The convulsions of a symmetrical pair of muscles, such as the two gastrocnemii or the two extensores communes digitorum, are of the same character and intensity. The contractions of the gastrocnemii, muscles much larger than the extensors of the forearm, are of course much greater than the contractions of the latter muscles, but there is, practically speaking, no difference in the contractions of the symmetrical muscles on the two sides.

3. The convulsions set in within from thirty seconds to two or three minutes after intravenous injection of the poison. They consist of a series, varying in number, of clonic, followed by tonic contractions. The first series, after a short period of relaxation, is frequently followed by another series, and this, in a similar manner, by another, each series having the same characteristics as the first. At the termination of the convulsive state the animal is quiescent, but now and then, even without the administration of an additional dose of the poison, another cycle starts up, similar to the first. The animal may collapse and die even before the appearance of the convulsions, or may live for a considerable time.

4. The myogram, which records the contractions of the gastrocnemii (flexors) and the contractions of the extensors, two pairs of muscles of an antagonistic function, also illustrates the law of reciprocal innervation of the antagonists as taught by Sherrington.[24]

24. Sherrington, C. S.: Integrative Action of the Nervous System, New York, 1906, p. 85; ibid., p. 282.

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The innervation of the flexors, whether through reflex activity or excitation of the cerebrum, is, according to this law, associated with simultaneous inhibition and relaxation of its antagonistic extensors; and vice versa, the innervation of the extensors with inhibition and relaxation of the flexors. The inhibitory effect in the latter instance, however, is not nearly so marked as in the first, owing to the fact that the flexors are more powerful muscles than the extensors, and their active innervation is accordingly associated with a more pronounced reciprocal effect on the latter muscles.

A study of the myogram shows that simultaneously with a clonic contraction of the gastrocnemii, there is a depression and relaxation of the extensores communes digitorum, and their levers go below the abscissae;[25] also that the contractions of the latter, when their levers rise above the abscissae, correspond with the relaxation of the gastrocnemii. This, as shown in the myogram, is repeated a number of times. Inhibition of the extensors is now and then noted (as shown in Figure 2) when the innervation of the flexors is not sufficiently intense to bring about their obvious contraction, a fact which has been observed also by Sherrington in his studies of inhibition through reflex excitation. The action of the oil of absinthe is thus essentially different from that of the spinal stimulants, such as strychnin or tetanus toxin which, as is well known, are destroyers of reciprocal innervation.[26]

These various phases of the absinthe convulsion should, I believe, in the light of well established physiologic facts, be explained as follows:

In accordance with the pharmacologic law of dissolution drugs acting on the brain (alcohol, ether, chloroform, morphin, etc.) first affect the higher cen-

25. It is, of course, understood that while the myogram records the contractions of the flexors of the hindlimbs, the flexors of the forelimbs, not recorded, are also in convulsions.

26. That the depression shown in one group of muscles during the contraction of the antagonistic group is due to reciprocal inhibition is proved by the fact that in myograms of absinthe convulsions in which contractions of the flexors exclusively were recorded (the gastrocnemii as the flexores sublimes digitorum), all the levers rise and fall simultaneously (Fig. 3).

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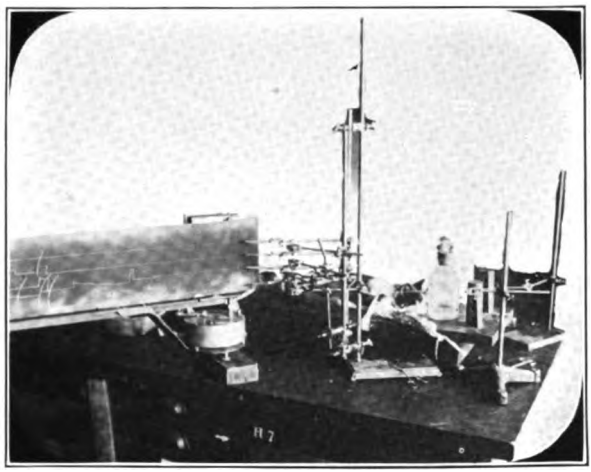

Fig. 1.—Apparatus for making myograms.

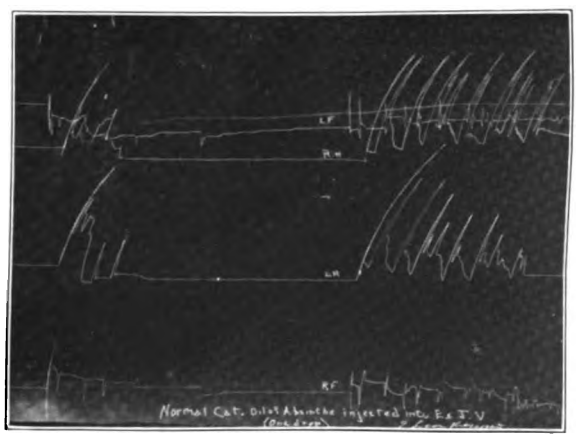

Fig. 2.—Myogram of an absinthe convulsion in a normal animal, based on a study of four normal cats.

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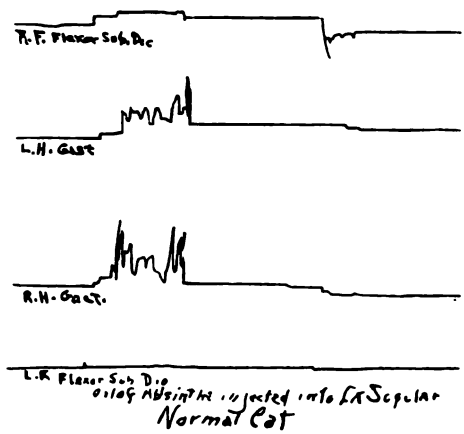

Fig. 3.—Myogram of an absinthe convulsion in a normal cat, in which the contractions of the flexors (of forelimbs and hindlimbs) exclusively were recorded.

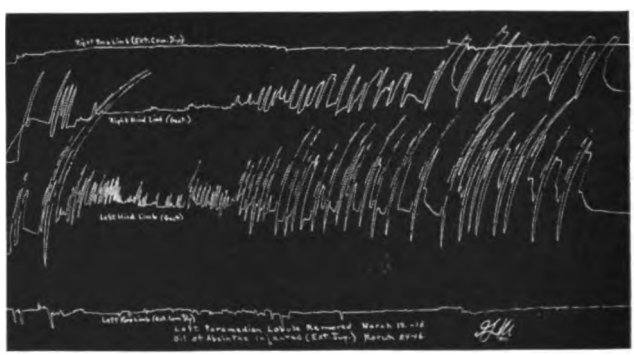

Fig. 4.—Myogram of a generalized absinthe convulsion in a cat with a circumscribed lesion of the left lobulus paramedianus.

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Fig. 5.—Brain of cat with lesion of left paramedian lobule (myogram, Fig. 4).

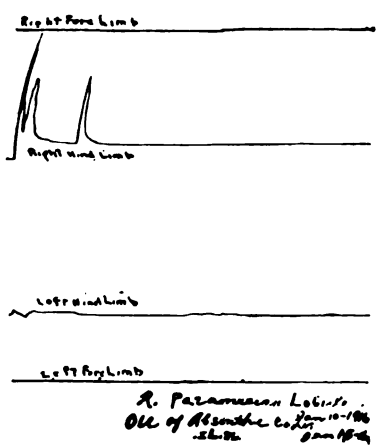

Fig. 6.—Myogram of a localized absinthe convulsion from a cat with a small lesion of the right lobulus paramedianus.

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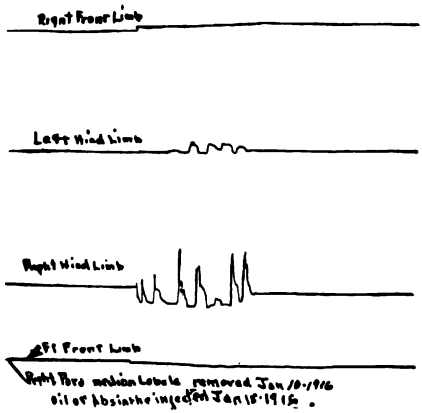
Right Front Limb
Left Hind Limb
Right Hind Limb
Left Front Limb
Right Pneumothorax Cannula removed Jan 10-1916
Oil of Absinthe injected Jan 18-1916

Fig. 7.—Myogram of absinthe convulsion from the same cat from which the myogram shown in Figure 6 was obtained, after the order of attachment of the gastrocnemii to their respective levers had been reversed.

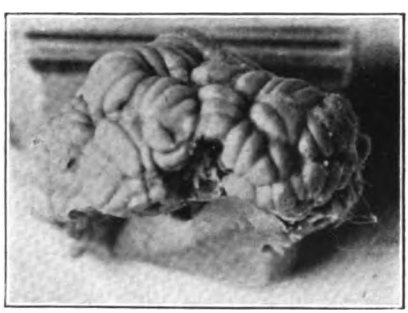

Fig. 8.—Myogram of a localized absinthe convulsion from the same cat from which the myogram shown in Figure 6 was obtained, the order of the attachment to the gastrocnemii to their respective muscle levers having been reversed.

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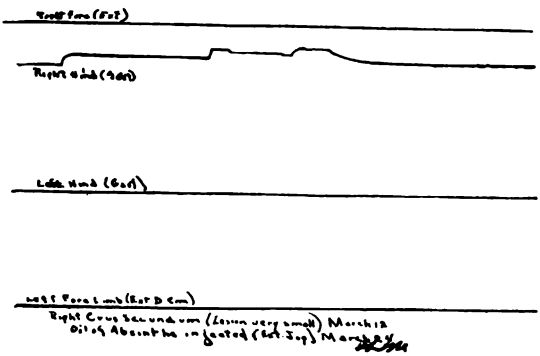

Fig. 9.—Myogram of a localized absinthe convulsion in animal with a lesion of the right crus secundum.

Fig. 10.—Brain of cat from which the myogram shown in Figure 9 was obtained. A similar tracing from an animal with a small lesion of the left crus secundum is shown on the drum in Figure 1.

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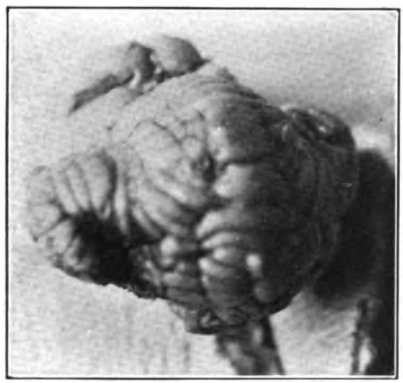

Fig. 11.—Brain of cat from which myogram shown on drum was obtained.

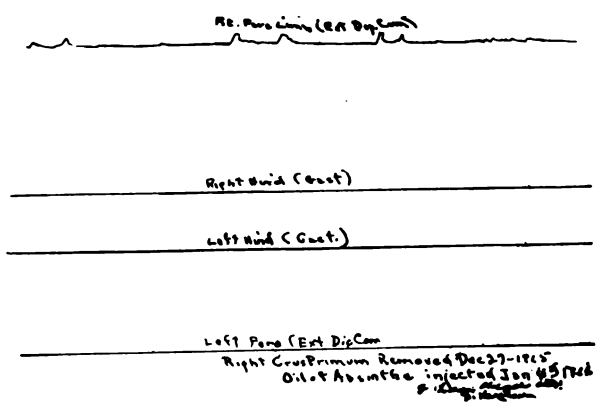
Rt. Pons Cinis (Ext Dig Com)
Right hind (Cont)
Left hind (Cont.)
Left Pons (Ext Dig Com
Right Crus Primum Removed Dec 27-1915
Dilut Absinthe injected Jan 4 5 1916

Fig. 12.—Result in an animal with a small circumscribed lesion of the right crus primum.

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Fig. 13.—Brain of cat with lesion of right crus primum.

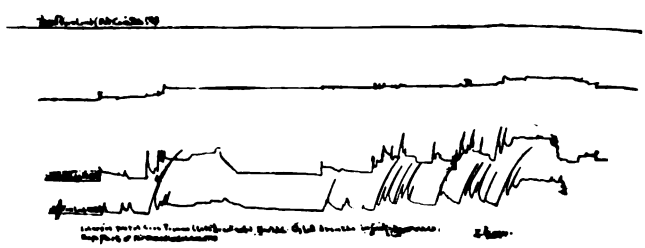

Fig. 14.—Myogram of localized absinthe convulsion of the left crus primum; the superior part of the left paramedian lobule was also involved.

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Fig. 15.—Brain of cat with lesion of median part of left crus primum and superior end of left paramedian lobule.

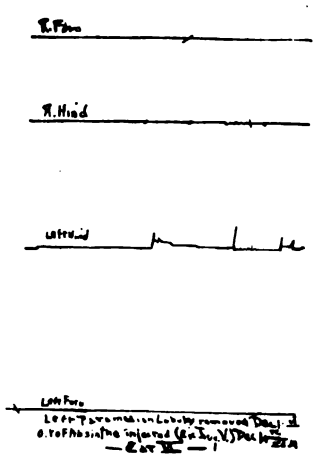

Fig. 16.—Localized absinthe convulsion from cat with lesion of left paramedian lobule (brain is not shown).

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ters, the centers which in the process of evolution are the last to attain maturity. In like manner, this poison exerts its influence first on the motor cortex of the cerebrum. The excitation of this part of the brain which, as described by Sherrington, predominantly innervates the muscles that are concerned in "transient, phasic" movements (or "changing" movements, according to Hughlings Jackson[27]) produces a clonic contraction of the flexors with a simultaneous inhibition of the extensors. The poison next affects the lower centers, those that in the process of evolution mature early, the tonus centers in the medulla, which predominantly innervate the extensors, the set of muscles employed "in steady, tonic action and maintenance of attitude" (Sherrington), or in "continuous movements" (Jackson). This produces a contraction, chiefly tonic in character, of the extensors with a simultaneous depression of the flexors. The depression in this case is not nearly so pronounced, however, as that which is associated with the contraction of the more powerful flexors. The inhibition of the extensors in the latter instance is so great that their levers fall below the abscissa, while that of the flexors, which results from the comparatively mild innervation of the weak extensors, is but slight and their levers remain above that line. These phases of the myogram are repeated until the effect of the poison wears off.

In view of these facts, a lesion of the crus secundum, for example, should, in accordance with Bolk's theory of cerebellar localization, and the view of the cerebellar function presented in my last communication, result, after intravenous injection of absinthe, either in a greater intensity of the convulsions, both clonic and tonic, in the muscles of the homolateral hindlimb, or in the presence of convulsions in these muscles while the rest of the body is quiescent. A lesion of the crus primum should be followed by similar results in the

27. Jackson, Hughlings: Med. Exam. and Pract., April 5, 1877; Med. Times and Gaz., 1878, ii, 485; Brain, London, 1906, xxix, 425.

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muscles of the homolateral forelimb, etc.[28] The results in my experiments, as shown in the myogram, any one of which is illustrative of all others in the same type of a lesion, are, in general, confirmatory of both these suppositions.

It will be noted that the contractions of the left gastrocnemius were far greater than those of the right gastrocnemius, whereas the contractions of the extensors were practically alike, clearly indicating a relationship between the lobulus paramedianus with the homolateral hindlimb.

A generalized convulsion in the animal operated on, with its low state of vitality, is, however, very difficult to obtain, owing to the fact that the oil of absinthe, which is a powerful narcotic poison, when administered in comparatively large doses, generally kills the animal without giving rise to convulsions. It was therefore thought preferable to administer the oil in very small doses and note whether convulsions would appear in one muscle group while the others remain unaffected.

A myogram of such a localized convulsion is shown in Figure 6.

It will be seen from this myogram that whereas both extensors were quiescent, both gastrocnemii were in contractions. The muscle on the side of the lesion contracted, however, much more vigorously than the corresponding muscle on the other side, this fact again indicating a relationship between the lobulus paramedianus and the homolateral hindlimb. To make certain that there was no mechanical difficulty present which might have prevented the recording of the left gastrocnemius and which I might have failed to observe visually, I reversed the order of the attachment of the two gastrocnemii to their respective muscle levers and took a myogram of another series of convulsions. The myo-

28. The convulsions resulting from intravenous injection of absinthe after complete removal of a lateral lobe have been studied by Risien Russell, who found them to be much more intense on the side of the lesion (Phil. Tr. Roy. Soc. Lond., clxxxv, 842). They were found to be identical on both sides by Rubert Boyce (Phil. Tr. Soc. Lond., clxxxvi, p. 370).

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gram (Fig. 8) shows the same characteristics as the preceding one.

The last two myograms are, with but two exceptions, typical of all the other records of localized convulsions in animals with a circumscribed lesion of the lobulus paramedianus, three of which had a lesion of the left lobule and one, a lesion of the right. It is characteristic that, during the convulsive state, whereas the extensors are perfectly quiescent, both gastrocnemii are in convulsions, the homolateral muscle being affected, however, to a much greater degree than the contralateral muscle. In the two exceptions referred to, the homolateral gastrocnemius was contracting while the contralateral muscle, as well as the extensors, were quiescent. This last type of localized convulsions was, however, the rule in the two animals with a lesion of the crus secundum.

It will be seen that here the right extensor communes digitorum was in convulsions although the rest of the body was completely at rest. Contractions of the muscle of the type shown in the myogram have been recorded throughout the whole extent of the drum. An identical tracing, with the contraction of the left extensor communis digitorum and the rest of the muscles quiescent, was obtained from an animal with a lesion of the left crus primum. A relationship is thus shown between the crus primum and the homolateral forelimb.

Figure 14 is of special interest. This myogram was obtained from a cat with a comparatively large lesion of the left crus primum as shown by the photograph of the brain, which careful examination has shown to have involved also the superior part of the left paramedian lobules.

The record shows that whereas the right extensor communis digitorum was perfectly quiescent, the left was in a state of intense convulsions, its contractions being much greater than those of the voluminous and powerful gastrocnemius. This condition is never

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observed in a normal animal. It shows also that both gastrocnemii were, at the same time, also in convulsions, the homolateral muscle contracting, however, much more vigorously than the contralateral one. The myogram thus represents the result of a combined lesion of the left crus primum and the left paramedian lobules.

The various myograms show the greater intensity of the convulsions in the muscles related to the site of the lesion exhibited itself in both their clonic and tonic contractions, but mainly in the first type.

In another animal with a lesion of the lobulus medianus posterior the oil of absinthe produced intense convulsions in the posterior of the trunk, although the muscles of the limbs were apparently unaffected. These convulsions were observed visually and I have no graphic record of them.

The lobules studied in these experiments have the following homologues in the human cerebellum: The lobulus paramedianus corresponds to the tonsil or amygdala, the crus secundum to the lobulus biventer, and the crus primum to the lobulus semilunaris superior et inferior and the lobulus gracilis.[2]

SUMMARY AND CONCLUSIONS

1. The function of the cerebellum is to inhibit, control and regulate the activity of the motor cortex of the cerebrum and the paracerebellar nuclei in the medulla.

2. The phenomena of cerebellar deficiency are, accordingly, to be interpreted as phenomena of hyperfunctional and not hypofunctional activity.

3. The cerebellum is functionally differentiated for the various muscle groups of the body, indirectly, by being primarily related through its various lobules to the various motor centers in the cerebrum and the tonus centers in the medulla, just as a posterior root ganglion is, in a motor sense, related to a certain muscle complex through its corresponding group of motor cells in the anterior horn of the cord.

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4. The paramedian lobule is, in this manner, related to its homolateral hindlimb, and probably also to the contralateral hindlimb; the crus secundum to the homolateral hindlimb, very likely, exclusively, and the crus primum to the homolateral forelimb.

5. These results are in general in conformity with the theory of cerebellar localization as postulated by Bolk. They differ from it only as regards the paramedian lobule, which Bolk assumed was the center for unilateral movements of the muscles of the trunk.

It might be well to state in this connection that this study might prove to be of distinct practical importance. It is possible that in suspected cases of cerebellar tumor or disease in which the phenomena of cerebellar deficiency, the ataxic gait, the hypermetria, the adiadokocinesis in the arms, etc., are too slight to be recognized, the administration within physiologic limits of a cerebral excitant, such as the vinous preparation of absinthe or even ordinary alcohol in moderate doses, might make these phenomena obvious and thus aid us not only in diagnosing an affection of the organ but also in establishing the exact seat of the disturbance.

In closing I wish to acknowledge my indebtedness to Prof. A. J. Carlson, of the University of Chicago, for giving me the privilege and facilities for carrying out this work in his laboratory and for valuable suggestions; and to Mr. D. Kaplan, a student of the university, for his able and conscientious assistance in all these experiments.

ABSTRACT OF DISCUSSION

DR. CHARLES K. MILLS, Philadelphia: Dr. Meyers in a general way takes somewhat the position which Dr. Weisenburg and I took with reference to the matter of cerebellar localization. He seemed to be distinctly advocating a localized control of certain movements through the control of specific cortical and other centers. The cerebellum controls in certain of its parts certain synergic movements. The elements of the synergic movements have differentiated centers in the cerebrum. The cerebellum in any of its centers does not control separately and distinctly an impulse which would cause such a movement of the index finger as flexion or extension, alone, but the particular duty of the cerebellum is to regulate that

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movement in so far as it may be associated with other movements. In other words, cerebellar centers represent more or less complex synergic movements. Crocq pointed out that the centers of tone are distinctly different in location in lower animals, and as we go up in the scale, as in the frog, the dog or cat, the ape, etc., until we reach man. The mechanism of tone in the human being is different from that for the lower animal, but man may be reduced to the lower animal scale by certain lesions, and become more or less automatic and reflex in his activities. In complete bilateral flaccidity due to a total transverse lesion of the cord and in hemiplegic flaccidity due to a total destruction of the internal capsule, tonectic impulses can no longer be conveyed to the limbs affected, although they may be received by the pyramidal system above the side of the lesions.

DR. TOM A. WILLIAMS, Washington, D. C.: Dr. Meyers seemed to make little of the clinical manifestations of alteration of tonus; and yet he attributes all the disorders expressing cerebellar lesions as due to interference with tonus. If that were the case, we ought to find clinically in cases where there are disorders of motility definitely established as due to cerebellar lesions at post-mortem, very marked interference with tonus. For example, is it conceivable that such marked interference with motility as occurs when there is a complete suppression of labyrinthine deviation from central lesion can be attributed to disordered tonus when there is no demonstrable disorder of tonus clinically? Therefore we should look a little farther perhaps than mere alteration of tonus for such marked symptoms as the loss of normal conjugate deviation on labyrinthine stimulation. Even supposing that such lesions do involve the paracerebellar nuclei, yet even there one should be able to demonstrate a difference of tonus on the two limbs of that individual who shows such symptoms. Will Dr. Meyers be more explicit regarding what he has said in relation to the objection? There are many other questions aroused by this research, but I think the factor mentioned is the most important.

DR. CHARLES K. MILLS, Philadelphia: The paracerebellar nuclei are, I believe, distinctly related to the distribution of the vestibular nerve, and they have special functions which have actually been demonstrated not only by the Bárány methods, but by other clinicopathologic investigations. The function of this nucleus complex is different from that of tonus in my interpretation of the word. And we must reconcile the reasoning which Dr. Meyers has advocated with the observations of other sort bearing on this subject.

DR. I. LEON MEYERS, Chicago: Dr. Mills expresses the belief that the cerebellum controls synergy of movement. My aim was not to deny the control of synergy of movement—it was to analyze synergy. What does synergy mean? As stated in my last paper, synergy is extensive work and can

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not be expressed in the terms of a single neuron effect. It implies that the whole cerebellum works at the same time. Nerve activity in a single neuron is limited, and could only act on one single muscle fiber. My idea was to show that the cerebellar function is that of coordinating movements in a manner similar to the coordination of movement in the heart by the inhibitory effect of the vagus. The voluntary movements of the skeletal muscles it seems are the result of two antagonistic forces: the cerebellum and the cerebrum, just as the activity of the heart, and probably also that of the plain muscles of the stomach, intestines, etc., are the resultant effects of two antagonistic forces, the sympathetic and the vagus. This resultant effect may be called synergy, but the primary activity is that of acceleration of movement, and inhibition of movement. Owing to lack of time I omitted one point. One or two tracings were introduced to show that following cerebellar lesions there is, in the absinthe fit, an increase also of the tonic contractions, indicating that the cerebellum checks also the impulses of tonus. This as stated in my paper, has been demonstrated before by Thiele by his studies of decerebrate rigidity. That the paracerebellar nuclei are the center for tonus stimuli has been held long ago by Ewald, who advanced the idea of labyrinthine tonus, the Deiter's complex being merely a station in the labyrinthine mechanism. But the chief action of the cerebellum seems to be on the motor cortex as shown by the tracings in which a much greater intensity is noted in the clonic than in the tonic contractions. I should also like to add that in several experiments the lesions were too small to be photographed, and no symptoms whatever were to be observed. On injection with absinthe, however, there was prompt reaction on the side of the lesion. With reference to the cerebellar localization it must be accepted in the same sense as Horsley viewed it—as sensory centers with their primary influence on the motor cortex, in the same manner as the ganglia of the sensory roots which pass into the lumbar region are centers with relation to the musculature of the leg.

DR. TOM A. WILLIAMS: Why is there no demonstrable difference of tonus in the patient then?

DR. MEYERS: In examining reflexes which are probably the expression of tonus, we find that they are variable. Sometimes they are increased, and cases have been quoted by Gowers in which they have been decreased. So I really think that no definite conclusions can be reached as regards tonus from a study of the reflexes in cerebellar lesions.

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EPILEPSY

WITH SPECIAL REFERENCE TO TREATMENT

FRANCIS X. DERCUM, M.D.
PHILADELPHIA

A disease as old as epilepsy is always interesting and constantly demands our best efforts for its alleviation and its possible cure. In order to approach its treatment intelligently, it is necessary first that we obtain as clear conceptions as possible concerning its nature. Let us, therefore, briefly review some of the essential facts.

It is at once evident that in a large number of cases the history of epilepsy begins before the individual is born, indeed, before the foundation of the organism is laid. All writers, ancient and modern, lay stress on the factor of heredity. Most writers content themselves with the general statement, others express the fact in percentages. Gowers, for instance, found hereditary factors at one time in 35 per cent., and at another time in 46 per cent. of his cases, Ahronson in 32 per cent., Siebold in 55.2 per cent., Dejerine in 66.8 per cent., Binswanger in 61.7 per cent., and Kraepelin in 45.7 per cent. An unusually low percentage is given by Petges and Cardenal, namely, 22 per cent. Differences in the estimates made by different observers are doubtless to be explained by differences as to what they have included under hereditary factors. The results, however, justify the conclusion that in a large number of cases these factors are present. If all forms of nervous and mental diseases in the ancestry are included, the percentage, of course, rises. If the inquiry is restricted to the transmission of epilepsy alone, the percentage, of course, falls. The results must further vary

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whether we include a general or collateral family history of epilepsy or whether we limit our studies to its direct inheritance. Again, Kraepelin found in 303 men 44.5 per cent. presenting a family history of nervous and mental diseases, but only 32.7 per cent. presenting such a history in the immediate parents of the patients. Further, when the inquiry was restricted to the occurrence of epilepsy alone, a general family history of epilepsy was found in 13.2 per cent., and its direct transmission from parent to child in only 6.3 per cent. In 104 women, Kraepelin found 49 per cent. with a general family history and 39.7 per cent. with a direct history of nervous and mental diseases; 17.3 per cent. with a general family history of epilepsy and 11.5 per cent. with a direct history. It would appear that a family history of nervous and mental diseases is of great significance, and this is indeed in accord with general experience.

Other facts must also be taken into account. Among these is alcoholism in the ancestry. That the children of alcoholics frequently suffer from epilepsy is well known. Kraepelin, for instance, found a direct alcoholic ancestry in 18.2 per cent. of 303 men and in 19.2 per cent. of 104 women. That there is here a direct damage of the germ plasm there can be no doubt. Again, the rôle which syphilis in the ancestry plays is also an important one. It is probable that syphilis acts much as alcohol does, namely, by damaging the germ plasm. In a given number of our cases, moreover, the actual evidence of syphilis can be demonstrated clinically and by the Wassermann reaction in the epileptic children. In other cases, while it cannot be asserted that the patients suffer from inherited syphilis, it is probable that the development of the organism has been hampered, arrested, and made deviate and degenerate by the presence of the spirochete and its toxins. In the larger number of cases, however, it is probable that the baneful effect of syphilis in the parent has expended itself mainly in an impairment of the germ

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plasm. It is probable that toxic factors in the ancestry other than those mentioned, such as lead poisoning and the various infections generally, occurring in the ancestry, may damage the germ plasm so as to lead to the production of epilepsy in the descendants; but of this our knowledge is as yet insufficient to speak definitely. However, that the damage to the germ plasm from the various causes mentioned is not theoretical but actual and real is proved by the signs of morphologic arrest and deviation so commonly present. Among these are the anomalies in the size and shape of the skull, the high and narrow palate, the anomalies of dentition, of the ears, of the digits and of the general development. We must remember, too, that the signs of arrest and deviation accessible to observation are merely surface indications of other and more fundamental deviations present in the organism throughout.

Approaching the subject from whatever point of view we choose, the inference is unavoidable that in a large number of epileptics there has been a primary, a basic impairment of the germ plasm. It would appear that this impairment may be general in character, the result of various nervous and mental diseases or of various intoxications and infections in the ancestry; or, the impairment may be special in character and may result in the special transmission of epilepsy.

Concerning the direct production of epilepsy in the individual himself by intoxications and infections, the evidence is overwhelming. This is notably true of alcohol. The sufferer from alcoholic epilepsy is the not infrequent subject of both outpatient and hospital care. Lead poisoning also plays a rôle here, but naturally a less important rôle. Regarding the infections and the rôle which they play in the production of epilepsy, the evidence is also quite conclusive. The convulsions which now and then accompany the infectious diseases of childhood are to be regarded merely as epiphenomena of the infectious processes, and are

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to be explained by a direct toxic action on the cortex. Quite commonly they disappear with the infection; they are of consequence only when they persist subsequently. Unfortunately, they occasionally persist as established epilepsies. Again, epilepsy every now and then follows an attack of typhoid or of scarlet fever, less frequently of measles, whooping cough or influenza. Not uncommonly there is a history of convulsions accompanying the infection, and then, after an interval, possibly of months or of years, the establishment of epileptic attacks follows. It is probable that in such cases an encephalitis, probably localized and cortical, occurred during the febrile infection, followed subsequently by sclerotic changes, perhaps slight in character, but sufficient to act as the starting point of future epileptic seizures. I have only recently studied a case of this kind, one in which an attack followed by weakness of one half of the body occurred during the course of typhoid fever, and in which some years later typical epileptic seizures supervened. The occurrence of epilepsy as a sequel of encephalitis the result of infection has been emphasized by Freud, Rie, Marie, Renlich and others.

Another factor in the history of epilepsy is that of trauma. Here again the facts are such as not to admit of dispute. Certain it is that cases are constantly encountered in the clinics, in which injury to the brain has been followed after a longer or shorter interval by epilepsy. Epilepsy following injury to the brain is frequently jacksonian in type, but it is not improbable that changes may supervene in the traumas of childhood which may later give rise to the picture of a generalized epilepsy. It is exceedingly probable that in the epilepsies which supervene after intoxications, infections and traumas, actual changes are present in the cortex, possibly of the nature of mild and limited scleroses themselves the sequels of limited encephalitis. Allied to such epilepsies are the epilepsies seen in hemi-

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plegic and diplegic children. Here atrophic and sclerotic changes more or less pronounced involve the motor area of one or both sides and serve as the cause of the convulsive seizures. It is significant in this connection that many epileptics are left handed; in these cases the left handedness is probably an acquired quality subsequent to a cortical left-sided damage in infancy or early life. In other cases, it is doubtless connected with some morphologic deviation. The epilepsies which are the result of coarse scleroses are in turn closely related to the epilepsies caused by neoplasms and gross lesions generally; these need not here detain us.

When we review the facts which have here been hastily summarized, one fact stands forth with striking prominence, namely, that epilepsy is not a specific clinical entity. Under the caption of epilepsy are included many symptom groups which differ widely as to their origin and pathology. Perhaps this is not surprising when we reflect on the physiology of the brain. It would appear that the motor area of the cortex responds by convulsive attacks to both chemical and physical irritants, and that there should be a multiplicity of causes, any one or number of which may bring about an epileptic symptom group, is perhaps just what we should have been led to expect.

The lesson taught by the pathologic findings at necropsy are also of great significance and in keeping with what has already been said. Many years ago, I placed on record anatomic studies of twelve epileptic brains all of which revealed more or less marked anomalies of the convolutions and fissures. These findings were all to be interpreted as phenomena of arrest and deviation. A similar interpretation is to be placed on the sclerosis of the cornu ammonis so much insisted on by earlier writers. Arrest and deviation are also the significance, as I have already pointed out, of the asymmetries, peculiarities and malformations of the skull.

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Microscopic studies of the brain have sometimes revealed atrophic changes in the cortical cells, and sometimes a proliferation of the glia, more especially of the cortex in its outer layers, a proliferation which constitutes a kind of gliosis or sclerosis. Time will not permit of an enumeration of the findings in epilepsy; the thickening of the calvarium, the thickening of the membranes, the adhesion of the latter to the skull or brain, or the visceral changes which necropsies have from time to time revealed. Suffice it to say that no changes are constant, and this is the factor of most significance.

If we attempt to classify the epilepsies, we encounter great difficulties. It will not suffice, for instance, to divide them into those attended by gross organic changes, such as tumors, massive scleroses, traumatic disease and the like, and those in which no such factors are evident, for many of the latter may, on microscopic examination, reveal the very changes which constitute organic disease. We have seen how minute and varied may be the changes and how varied the causes. Moreover, it is especially in the group without apparent gross organic cause that hereditary factors play such an important rôle, and in many of this group, again, morphologic arrests and deviations are present which in turn give to the cases a profoundly organic character.

Some epileptics present symptoms suggestive of involvement of the internal secretions. That such symptoms should occasionally be present is not surprising when we reflect that anomalies of the glands of internal secretion are frequently closely bound up with arrest and deviation. No special internal symptom group is presented; occasionally there are modifications of stature and growth; coarseness, dryness, thickness or suspicious infiltration of the skin and, less often, special signs distinctly referable to individual glands are observed. In quite a number of epileptics I demonstrated years ago by Roentgen-ray

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examinations enlargement and distortion of the pituitary fossa due apparently to disease of the hypophysis.

Once more let it be emphasized that it is just in the group often embraced under the term "essential" epilepsy that we have to deal with persons in whom the development has taken place in an aberrant and damaged germ plasm, and in which the epilepsy is expressive of an endogenous, autotoxic deterioration. The evidence before us is such as to exclude specificity of cause. I am, of course, aware of the recent announcement by Reed of Cincinnati of the discovery of a germ as the cause of epilepsy. If the correctness of his observations be admitted, it must be equally conceded that they can bear but a limited relation to the great and varied group of diseased states embraced under epilepsy.

This consideration as to the nature of epilepsy has unfortunately occupied more time than I had originally intended, but an intelligent discussion of treatment is impossible without it. Clear ideas must obtain, first, as to the conditions which confront us and, secondly, as to the objects which we should seek to accomplish. A specific treatment of epilepsy is obviously out of the question, and the first step must be the intensive study of each individual case. This includes an elaborate study of the family and of the personal histories, a detailed neurologic and visceral examination, including, of course, the laboratory tests of the blood and at times of the cerebrospinal fluid, and, finally and especially, a search for the earmarks of arrest and deviation. The latter, it must be remembered, are not only physical but also mental. That the treatment must necessarily be influenced by the findings follows of necessity; especially is this true when the investigations reveal gross organic nervous lesions, visceral disease or active infectious processes. What shall we do, however, in the residue of cases in which the investigation reveals merely the defective, autotoxic and deteriorating organism outlined in this paper? The task is always

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difficult, often discouraging and at times hopeless. However, not infrequently much may be accomplished and, in a few cases, even definite arrest of seizures brought about.

The first indication is that the organism, though defective and deviate, shall lead as physiologic a life as is compatible with its structure. To attain this end, a life without physical or mental strain, close to nature, in camp or on the farm, should be adopted by the epileptic. This, indeed, is the principle applied in the various epileptic colonies. In a given number of cases it is attended by an improvement in general health and a notable diminution in the number of seizures. There can be no doubt that the benefit is largely due to the increased oxidation of waste and toxic substances and the general increase of physiologic efficiency which result from an outdoor life. In addition, three points should be borne in mind:

1. The diet should be so modified that in this organism, already toxic, as little strain as possible be placed on the liver, the thyroid and other defensive glands. For this reason the red meats are to be partaken of sparingly. The carbohydrates also are to be diminished. To take the latter in large amount is to hamper the oxidation of the tissues, an oxidation which for the obvious reason of the autotoxicity of the patient should be maintained at as high a level as possible. In the diet, emphasis should be laid on the white meats, the succulent vegetables and milk; eggs also may be permitted. Stimulants of all kinds are, of course, to be excluded.

2. The various avenues of elimination should be kept freely open. If the diet does not of itself counteract the constipation frequently present, a moderate dose of a simple saline or laxative water may be given daily. The patient should drink water freely between meals to promote the action of the kidneys, and should take a lukewarm sponge bath daily to promote the

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action of the skin. The bath should not be such as to promote an active reaction, but merely to favor elimination.

3. Resort to medicines must, of course, be had in many cases to influence or control the seizures. Time will not permit the extended discussion of these, but after all is said and done, experience teaches that chief reliance must be placed on the bromids. Regarding their efficient administration, however, one important point must be borne in mind, namely, the principle of sodium chlorid withdrawal introduced by Richet and Toulouse. If table salt is withheld, the bromids instead of being eliminated are retained and are effective in much smaller dose. I myself have been in the habit for many years past of administering the bromids in the form of sodium bromid, at the same time instituting as rigid a withdrawal of the sodium chlorid as possible. There can be no doubt that under these circumstances the sodium bromid takes the place, in a measure, of the sodium chlorid in the tissues. If, in a case so treated, the sodium bromid be discontinued and sodium chlorid resumed, the bromid is rapidly eliminated in the urine.

But one other point of importance remains. In a given number of instances, the physiologic level of the patient may be distinctly raised by the administration from time to time of small doses of thyroid extract; say from an eighth to a quarter of a grain three times daily, seldom more. Thyroid in small doses, long continued, stimulates the chain of glands of internal secretion, increases oxidation and promotes metabolism generally.

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ABSTRACT OF DISCUSSION

Dr. David F. Weeks, Skillman, N. J.: I want to emphasize the importance of early recognition of symptoms that point toward beginning epilepsy. When the patient reaches the institution the disease has usually been of long standing. In the collection of data bearing on the heredity of patients at

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the New Jersey State Village for Epileptics at Skillman we were impressed with the importance of careful study of children subject to periods of unprovoked anger, joy, passion, weeping, etc., and accordingly undertook a special study of children of school age. For the purpose of checking our data our field workers investigated the heredity of children showing these epileptic characteristics and tendencies and reported the presence of the epileptic taint in many of these families. In some cases these potentially epileptic children later developed epileptic convulsions. School inspectors have an excellent opportunity of detecting these children before their seizures have been established, and by referring them to the family physician for such care and treatment as will put them under right living conditions may forestall the convulsions and reduce the number of epileptics. Our special classes should be reorganized to provide separate classes for epileptics, thus removing them from the classes as now established.

Dr. A. L. Skoog, Kansas City, Mo.: In connection with institutional or colony cases and cases seen in private practice, the point that strikes me in particular is the difference between the two classes of patients. It is true that a large percentage of the cases seen in colonies are of the chronic type. Their etiology is more obscure than that of the cases seen in private practice. Permanent damage has been done, and the etiologic factor or the immediate cause of the trouble is probably no longer at hand. I believe that the prognosis is better for cases observed in private practice than for those in colonies, for two reasons: First, they come at an earlier stage and you have better opportunity for a study of the etiology, and second, you can carry out certain treatments better. However, there are certain types of epilepsies that never should be taken care of in private practice. Such patients should be sent to a special colony for care and treatment. I like very much the way Dr. Dercum handled the subject from the standpoint of calling the condition *epilepsies,* not epilepsy. I believe it is now almost the universal opinion of students of this broad subject that we are dealing not with a single entity, but with a whole group of diseases involving the brain directly or indirectly. I believe it is possible that quite a number of epilepsies have their origin in mild or more severe degrees of encephalitis, secondary to one of the various infectious diseases of children. It is true that a large number of epilepsies are still described under the idiopathic or genuine epilepsies, so called, and we can say regarding these that the etiology is obscure. Some of these cases may have their origin in functional disturbances. This also has a bearing on some of the findings observed at necropsy. Most cases of epilepsy come to the postmortem table at a late stage. Every seizure produces at least a small amount of

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damage to the brain. I have had opportunity of observing three generalized convulsions in epileptics at operations, two of them in the parietal or postrolandic region, and one in the frontal region close to the head centers. Two of the convulsions were produced by bipolar electrical stimulation. There was nothing focal about the convulsions in either one of these cases, being diffused immediately. But the essential point was the tremendous amount of intracranial pressure produced. The surgeon with whom I was working at the time became very much alarmed, fearing serious damage from bulging of the brain. As soon as the convulsive period was over, not occupying more than one and one-half minutes, the brain immediately receded. A condition like this undoubtedly presents the possibility of trauma to the cortex, and that trauma may increase the susceptibility to future seizures.

DR. FRANCIS R. FRY, St. Louis: I do not know how much other practitioners and particularly neurologists have been disturbed by a phase of so-called treatment now in vogue. We certainly find it a disturbance, because 50 per cent. of the epileptic patients who come to us inquire whether or not they shall go over to Cincinnati. Some extreme thing of this kind obtrudes itself from time to time, and particularly is this true in regard to chronic affections and often in regard to epilepsy. Therefore it is very timely at junctures of this sort that there should come forth some authoritative statement such as the paper that has just been read. Dr. Dercum has conscientiously told us all that is known to date about epilepsy from any scientific or rational, sensible standpoint. And among practitioners, at least, it is something definite to appeal to, coming from one of Dr. Dercum's standing.

DR. CHARLES R. BALL, St. Paul: I was much pleased that Dr. Dercum brought out the fact that epilepsy is composed of separate entities and is not a single distinct disease. It may come from a great number of causes. The cause may be mechanically irritative, from any organic disturbance in the brain; or it may be chemically irritative, due to a toxic condition. In this connection it is well to take into consideration the secretions of the ductless glands and the toxemias that they undoubtedly cause through irregularities of function. With reference to heredity and the influence that alcohol plays, might not the special influence of alcohol on these glandular structures be an explanation for the frequent occurrence of epilepsy in the offspring of alcoholics? I am continually impressed with the very close relationship which seems to exist between the so-called idiopathic epilepsy and migraine. Not only have I found migraine in the mother with epilepsy in the child or a reversal of these conditions but in the same patient the epileptic seizure being converted into a migraine. I have also seen migraine develop into epilepsy.

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We should attempt to do more for these patients. Anything suggested is worth while. I believe I have found something that is an improvement over the old bromid treatment, viz., luminal. It does not have the bad influence that the bromid has. In the great majority of instances patients can take it over a long period of time without untoward consequences. Once in a while a patient develops a rash or dermatitis. Luminal is chemically similar to veronal, except that the ethyl group in veronal is replaced by one phenyl group. I give ⅔ to 1 grain of luminal three times and sometimes four times a day. While I am not able to compete with the Cincinnati clinic in the number of patients as yet, nevertheless I find my cases increasing in number.

Dr. William Ravine, Cincinnati: Dr. Dercum alluded to the reported finding of a specific organism in epilepsy by Dr. C. A. L. Reed. Dr. Wherry, bacteriologist of the Cincinnati General Hospital, did not find the specific organism, neither did his animal experimentation corroborate the findings of Reed; the organism did not fulfil all the requirements laid down by Koch. We in Cincinnati are not so very proud of the operation of "short-circuiting" for epilepsy. It is a far cry from intestinal stasis and its treatment to the cure of epilepsy. A great number of these afflicted individuals have come to Cincinnati. This procedure is quite an extreme measure to resort to in the treatment of epilepsy.

Dr. Julius Grinker, Chicago: Many of these forms of epilepsy are the result of lesions acquired perhaps early in childhood, but not discovered until long afterward, when the epileptic attacks have become thoroughly established. The real etiology of epilepsy is still to be discovered. It is true we have given bromid for many years, and we have not cured our patients, although many of them are kept at work, have become self-supporting, and in spite of the fact that they occasionally have an attack of epilepsy, they are useful members of the community. If we had not treated them medicinally, as some would have us do, and had experimented with the operative procedures which originated with gynecologists, many of these patients would have been lost and many others would have become invalids. Regarding the drugs to be used: The bromids are, after all, our main medicine in the treatment of epilepsy. There are patients, however, who cannot take bromids for even a short time without developing acne and pustular eruptions, and bromidism even after small doses. In some instances I have been handicapped in the management of epilepsy by the fact that patients could not even take a 10-grain dose of bromids. For this reason I have resorted to the use of luminal in some cases and the results obtained were very favorable. Many patients who could not take bromids well could take luminal in doses of 1½ grains once a day; others needed two doses

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daily; very few needed three doses of 1½ grains a day. There is one point in connection with the administration of luminal that I wish to emphasize: Having given luminal, do not change to bromids, because they become ineffective.

Dr. R. A. Greene, Palmer, Mass.: If there are no specific epilepsies, why is it that in institutions for the insane and feebleminded there are so many cases with exactly the same heredity, exactly the same lesions, and they do not have epilepsy? If there is not any specific epilepsy, why is it that some of these absolutely organic conditions such as the pronouncedly feebleminded and those with hemiplegia and all sorts of organic lesions, who are mental equals and look like our epileptic patients, do not have convulsions? Must there not be some specific cause of epilepsy? I do not believe that there is any micro-organism to be described, such as the *Bacillus epilepticus*. I can conceive that all epilepsies must have a primary status or causative factor in some metabolic abnormality or internal secretion that is not functioning properly.

Dr. James H. McBride, Pasadena, Calif.: There are of course many forms of epilepsy, and various causes: the epilepsy of traumatism, toxemia, peripheral irritation, etc. Two cases of epilepsy due to peripheral irritation that I recall are I think worthy of a brief mention. A woman at 30 developed epilepsy during confinement with her third and last child. She continued to have two or three fits a year until the time that I saw her when she was 63. On examination I found very large and badly ulcerated internal hemorrhoids. They were removed and she has not since had any recurrence of the fits, having now been entirely free from them for eleven years. A young man of 20 developed epilepsy while in college and during six months had two violent attacks of grand mal and many minor attacks. I found he had very large internal hemorrhoids which were removed. This was seven years ago and he has not had any recurrence of the fits. As a matter of pathologic interest I may mention the case of a man who was shot in the left eye at the battle of Gettysburg in 1863. He stated that he soon after developed epilepsy and up to the time of his death in 1889 had a discharge from the left ear.

On necropsy I found the bullet lying on the temporal bone just over the middle ear: It had entered at the orbit, passed to the temporal lobe and gravitated to the bone below, where it had produced an abscess and necrosis of the bone by which the abscess had discharged into the middle ear and perforated the tympanum. The bullet which I removed in three pieces had remained in the skull cavity for twenty-three years.

Dr. Joseph M. Aikin, Omaha: I desire to make an inquiry regarding the relationship between migraine and epilepsy. In taking the history of patients complaining of epilepsy, I am

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impressed with the frequency of epilepsy, where there is a family history of migraine. I have wondered if the pathogenesis of migraine and epilepsy may not be practically the same, whether it be a specific germ or whatever the cause. I have in mind a young man whose history was that he had sick headaches until 17. At the age of 27 he came to me seeking relief from epilepsy, alleging that his epilepsy began at the age of 17, at which time his headaches ceased, and that his epileptic attacks had supplanted his headaches for ten years. Another case is that of a boy 8 years old who had a family history of migraine, suffered severe attacks of migraine till 14 years old, when without warning he had an epileptic convulsion. For six consecutive months he had on an average two or three epileptic convulsions a week, but no sick headaches. At the expiration of the six months the epileptic convulsions ceased and for eight years past he has what he had formerly, sick headaches. The relationship between the two conditions leads me to think of a toxemia of some kind, whether of endogenous or exogenous origin I do not know.

Dr. William J. Hickson, Chicago: No one has mentioned the association of hysteria with epilepsy, and perhaps I should take that fact as evidence of its not being of significance. My cases are sent to me by the probate court, and I have made some detailed studies of the relationship of hysteria to epilepsy in certain cases. I am sure that I have done the patient, his family, and his community a real service by directing treatment to the hysteria and then turning the patient back with his small remnant of epilepsy.

Dr. Francis A. Ely, Des Moines, Iowa: Fortunately or unfortunately I have been recruited from the ranks of the general practitioners into those of the neurologists. I have had the opportunity of observing a great many instances of head trauma in children, and have attended a considerable number of difficult confinements. I am impressed with the fact that we should attach a great deal more importance to head trauma as a factor in the production of epilepsy. Since the Roentgen ray has come to our service it has been interesting to note that many instances of apparently slight head trauma in children have shown definite skull fractures and serious head lesions. We are all familiar with the great compressibility of the head of the child in coming through the birth canal, and those who have had any general practice will remember many instances in which infants have been very difficult to resuscitate. They will also perhaps remember many others in which, a week or so following birth, temporary epileptiform manifestations have occurred. I believe that in many instances such children in later life become epileptics, and I simply make this observation in order that we may not forget that head trauma both in the infant at

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birth and subsequent thereto are probably very important factors in the causation of epilepsy, and that there are many so-called idiopathic epilepsies which are indeed of traumatic origin.

There are undoubtedly a great many cases of encephalitis which escape the observation of the general practitioner and yet if these patients were subjected to a critical examination at the time of their illness, definite encephalitic symptoms might be observed. This type of cases also undoubtedly increase our list of those ascribed to unknown or idiopathic influences.

Dr. C. Eugene Riggs, St. Paul: The pessimism of the profession with regard to the treatment of epilepsy has had a disastrous effect on the laity and consequently they have been willing to take hold of any will-o'-the-wisp that presented itself. Last month, at the meeting of the American Neurological Association in Washington, attention was called to the use of extract of pancreas in the treatment of epilepsy. Epileptics usually have a high blood pressure, which is indicative of epinephrin in excess. Pancreatin neutralizes this excess. It should be given in 5 grain doses, after meals. It certainly has a decided influence in lessening the frequency of the convulsive seizures. It has been in use such a short time that no statement can yet be made as to definite results. It should be given a fair trial.

Dr. I. Leon Meyers, Chicago: Epilepsy has from time to time been ascribed to structural changes in the brain. As a matter of fact, however, there is a striking difference between idiopathic epilepsy and jacksonian epilepsy. In idiopathic epilepsy there is practically always a loss of consciousness or at least a clouding of consciousness, whereas in jacksonian epilepsy consciousness is frequently retained. This shows that idiopathic epilepsy very frequently originates in parts of the brain other than the cerebral cortex. We know that we can remove the entire cerebrum without complete loss of consciousness; the loss of consciousness in idiopathic epilepsy would therefore point to an etiologic factor in the more vital centers of the brain. We are often impressed with the similarity between toxic epilepsy and idiopathic epilepsy. I had occasion to observe one case of idiopathic epilepsy in which the patient had typical tonic and clonic convulsions a short time after I had seen the convulsions in an animal on an injection of oil of absinthe and I was struck by the great similarity between the two. This would speak in favor of the toxic origin of epilepsy. It is well known that in producing convulsions in animals by the administration of strychnin or any of the other convulsants, if you touch the fur of the animal it promptly goes into convulsions. All that is needed to produce a powerful convulsion-reflex is the application of some kind of stimulus. This will explain the

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cases in which it has been noted that slight irritations produced attacks of idiopathic epilepsy. If the body is saturated with a poison having toxic properties similar to those of strychnin or absinthe, then a slight external stimulus is sufficient to produce generalized convulsions. This is evidenced also by the aura. It seems that there is some kind of poison in the blood affecting the lower level centers, and a slight stimulus which manifests itself by a certain aura brings about the generalized convulsions. But unconsciousness supervening immediately, the aura is cut off and the patient's knowledge of the stimulus is vague and indefinite. From my observations of convulsions in animals as well as in men, I believe that idiopathic epilepsy is toxic in origin, and not due to organic changes in the brain. Of course, organic changes in the brain may cause convulsions, but they are not, I think, the cause of genuine idiopathic epilepsy, and I heartily concur with Dr. Dercum who spoke of epilepsies and not epilepsy.

Dr. David S. Booth, St. Louis: It is quite evident that there are a great many causes of epilepsy, but many of them are toxic and I have no doubt that a large number of these result from toxins in the alimentary canal, especially in the intestines. A number of years ago Professor French of the Southern Illinois Normal University discovered an intestinal parasite which he called the *Gastropholus epileptalis,* and which he thought was the cause of many cases of epilepsy, hence the name given to it. However, the examination of the feces of, I believe, over a thousand cases revealed only a few infections with this parasite. So Professor French decided that this was not the cause of all the cases of epilepsy. Epilepsy may be due to rectal trouble, as I have noted in several cases. A case under observation was that of a physician in whom epilepsy was cured without any medicine whatever on the successful treatment of a rectal ulcer. As to treatment, parathyroid extract has not been mentioned, and I would like to know whether calcium has been used to any great extent. I have used calcium bromid with some good results, but perhaps no better than have been secured in some other cases not treated with this agent.

Dr. Robert McGregor, Saginaw, Mich.: I would like to ask Dr. Dercum if he has ever used, or heard of the use of, cerium oxalate in epilepsy. Usually the addition of strychnin or digitalis to a bromid mixture will lessen the amount required.

Dr. J. Matthew Pulliam, Fort Wayne, Ind.: I believe our main trouble is that the general practitioner throws up his hands in horror when an epileptic walks into his office, saying that there is no treatment. In my opinion the principal thing in the treatment of epilepsy is elimination, and I am not afraid of sodium chlorid and I do eliminate with sodium

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chlorid, giving a hard pressed normal saline tablet by mouth so that it will dissolve slowly in the stomach. I give with this a tablet composed of ⅛ gr. aloes, 1/40 gr. extract cascara segrada, 2½ gr. each of bicarbonate of soda, bicarbonate of potash and magnesium sulphate six times a day with a full glass of water. Just try it and see what the elimination is. That of itself will reduce the number of convulsions. There is nothing equal to bromid to stop convulsions. In looking over my records for the past ten years I find forty cases of epilepsy, thirty-two of which have had no attacks during a period ranging from two to eight years.

In regard to treatment with bromid, there is one point which may be applied to the dosage of bromid. Watch the blood for the hemoglobin content. Give the bromid until you have a drop in hemoglobin of 10 per cent., and you will find a drop in the hemoglobin content if you go high enough. Then cut the dosage in half, giving two weeks and intermitting two weeks. I accidentally discovered this fact nine years ago and have treated every case along this line. Up to this time I have not made any mention of it, not knowing whether the point was a practical one or not, as I have had but forty cases in which I could observe the effect. In the state institution at Logansport and Government Hospital at Washington, D. C., we had hundreds of cases, but all beyond the stage where we could do anything. Every one has neglected to treat the child. A girl came to me yesterday, aged 22, who at the age of 4 years had epileptic convulsions. A doctor treated her and her convulsive seizures ceased. On coming to womanhood she again developed epilepsy. The seizures started again when, because of development, extra work was put on the nervous system. Now I am giving bromid as I give iodin, in gradually ascending doses with remission, giving it two weeks and stopping two weeks. I take no account of a convulsive attack—it is not the cause, it is only the result. A toxic process is at the bottom of the trouble. Any man giving bromid without the promotion of elimination is wrong, although it does help for a time. So far as bromism is concerned, it is a very easy matter to prevent this. In regard to the skin eruption, Fowler's solution is specific. Give it in ascending doses in connection with bromid and you will not get much bromism.

Dr. Foster Kennedy, New York: We have had so many dogmatic assertions in this discussion of epilepsy that perhaps it would be well for us here and now to confess that we do not know anything definite about the etiology of this condition. The best proof of our ignorance is that so many different theories are put forward and accepted. From the West we hear of the epileptococcus and the removal of the large intestine, and from the East we are told that epilepsy represents an effort of the individual to resume his fetal

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position in the mother. All these various propositions are put forward perfectly seriously by speakers. While we can do a good deal to alleviate and to investigate, it is well at the outset, in order to reach the truth, to clear our minds of any delusions that we know much more than did our forebears about this subject.

Dr. Walter Timme, New York: We cannot treat epilepsy as a disease; we have to treat the epileptic as an individual. Therefore, in one case we get results with sodium chlorid, while with another we do not. Another point is this: That the introduction of the endocrine glands into the therapeutics of epilepsy must be done guardedly. You cannot give thyroid constantly as you would bromid, without endangering the balance of the internal glandular system and perhaps converting an epilepsy into, or even adding to an epilepsy, a glycosuria or an exophthalmic goiter, both of which contingencies have occurred. You cannot give a patient thymus, nor can you give a patient suprarenal gland, forever, without disturbing the same balance. You must give first one, then the other, or a third, or none. You may give one of the glands for a week; it may be necessary to give it for a month, but each patient must be the criterion of the treatment. And unless you do that you are bound to fail. Two points to show the close interrelationship between the endocrine glands and epilepsy might be cited, as follows: There is a seasonal variation in the epileptic attack. Seizures are always more severe and frequent in spring; also in the spring there is an increased thymus activity and an increased pituitary activity. Another point is that at those critical periods of life, such as the menopause, such as puberty, epileptics either are cured spontaneously or their troubles spontaneously get worse. And it is at just those periods also that the endocrine glandular system is called on to exert itself to its utmost. There is a parallelism, therefore, between disturbances in the endocrine gland system and apparent spontaneous changes in the epileptic seizures.

Dr. L. Kauffman, Buffalo, N. Y.: The question of calcium in the treatment of epilepsy has been touched on. Colloidal chemistry has a very strong bearing on the metabolic condition of the human body. Dr. G. H. A. Clowes, chemist to the New York State Cancer Institute at Buffalo, finds that there is a certain balance which the body normally maintains between calcium, on the one hand, and sodium on the other, and that as soon as you disturb this balance one way or the other there is trouble which may manifest itself in spasm, as tetany, etc., and epilepsy can likewise be considered one of the forms of disease ensuing. With Dr. James W. Putnam of Buffalo I have employed calcium with considerable success. Of course, we must first take into consideration elimination,

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the various causes of irritation must be removed, and all the other possibilities taken care of; if then metabolism is the factor, try calcium lactate. It is a better drug for the purpose than calcium chlorid. It does not irritate the stomach, and you can use 30 to 60 grains three times a day in a glass of water over a long period, but I think it better to use it over short periods. We have had favorable results and complete cures in cases so far studied.

Dr. George A. Moleen, Denver: In the treatment of epilepsy so many things have been advocated that conservatism and suspended judgment are demanded. The relation of hysteria to epilepsy is, I believe, showing a very perceptible narrowing of the gap. I would reserve my opinion of luminal, which has been used so largely in German clinics, because of its very close relative veronal, for we are seeing increasing numbers of habitués of veronal now. Therefore, it would seem that the same judgment should be applied to the use of luminal as would be applied to the routine use of opium.

Dr. F. X. Dercum, Philadelphia: I have been impressed by the consensus of opinion with regard to epilepsy, namely that epilepsy represents a multiplicity of conditions of which the convulsive seizures are merely epiphenomena. We should entertain clear ideas with regard to the principle of salt withdrawal as originally advocated by Richet and Toulouse. Salt is not of itself especially harmful to epileptics but during bromid administration salt withdrawal should be practiced as thoroughly as possible, for the reason that the bromid, in a measure, replaces the sodium chlorid in the tissues and is retained in the tissues for a much longer period than otherwise. In a case in which sodium chlorid has been withdrawn for some time and the sodium bromid freely administered, the sodium bromid will be eliminated rapidly in the urine when sodium chlorid is again taken, showing that there is an interrelation between the sodium chlorid and the sodium bromid in their retention and elimination. I could not, of course, in my paper enter into a discussion of the various other drugs which are being used in epilepsy. We are not infrequently forced to abandon the bromid and to try other remedies. I have also had a rather favorable experience with luminal. I have not, however, used it over very long periods of time because I have been afraid of establishing a habit. In regard to Dr. McBride's case, we all recall the fact that trauma, surgical operations and incidental infections, such as typhoid, frequently inhibit epileptic seizures for a greater or longer period. It would appear that the causes which modify the metabolism of the body for the time being influence the frequency of the seizures. Operations on the intestinal tract, not infrequently performed at the present day, such as gastro-enterostomy,

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excision of the colon, etc., recall to our minds vividly the sad experiences of the profession years ago with regard to the influence that pelvic surgery was supposed to have on hysteria, epilepsy and other functional nervous diseases, an experience which should have convinced us as to the futility of such measures. Again, shall we experiment on our patients with poisons, autotoxins and what not, in diseases which are essentially morphologic and neuropathic in their character? Sanity demands that we should now and then review the knowledge which our ancestors and we ourselves have so painfully acquired in order that we should not allow ourselves to be deluded into wild and inexcusable undertakings.

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THE PROGRESSIVE TORSION SPASM OF CHILDHOOD (DYSTONIA MUSCULORUM DEFORMANS)

A CONSIDERATION OF ITS NATURE AND SYMPTOMATOLOGY *

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The subject which I would present for your consideration is that curious form of motor disturbance known as the progressive torsion spasm or dystonia musculorum deformans.

Dec. 17, 1910. Ziehen,[1] at a meeting of the Berlin Psychiatric Society, demonstrated a child 10 years of age with a spasmodic affection of unusual type. Four similar cases had come under his observation, three in members of the same family.[2] In all, the affection had been gradually progressive and was characterized by spasm and hypertonicity of the musculature, with curious twisting and torsion movements of the extremities and trunk. The muscular spasm was considerably increased by active movement and diminished during rest. The gait and station were chiefly affected and there was marked lordosis of the spine. The tendon reflexes were present but difficult to elicit, because of the tension and torsion of the muscles. Sensation and intelligence were not affected, and, in one of the cases, necropsy had revealed no essential lesion. All of the patients were Russian Jews.

* From the Neurological Wards of the Montefiore Home and Hospital.

1. Ziehen: Tonic Torsions Neurose, Neurol. Centralbl., 1911, xxx, 109; Allg. Ztschr. f. Psychiat., 1911, lxviii, 281.

2. Schwalbe had previously recorded this group of cases from Ziehen's Clinic as "Eine Eigenthümliche tonische Krampfform mit hysterischen Symptomen," Inaug. Diss., Berlin, 1908.

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In discussing the character of the motor disturbance, he stated that while there was a certain general resemblance to the movements of chorea and athetosis, these affections as well as hysteria could be definitely excluded. He regarded the affection as a special type of the degenerative spasmodic neuroses, allied to the impulsive tics, and proposed the name "tonic torsion neurosis."

In 1911, Oppenheim[3] followed with a more elaborate description of the disease, which he regarded as organic and not functional in nature, and emphasized the close relationship of the spasm to athetoid movements.

Oppenheim's cases, like those of Ziehen, began in childhood (from the eighth to the fourteenth year). All were either Russian or Polish Jews. The affection began insidiously in one of the extremities, either the leg or the arm; but eventually the muscles of the thigh, pelvis and spinal column were always chiefly affected. There was marked lordosis with clownlike, bizarre movements, and a curious disturbance of the gait which suggested such descriptive terms as the "dromedary gait" or "monkey gait." In some of his cases backward progression was easier than the usual method. He also observed a tendency to clonic and rhythmic movements, mobile spasm and even tremor, somewhat resembling that of paralysis agitans. There was also a certain resemblance to chronic chorea, and still more to athetosis. The tendency to tonic muscular spasms with twisting and torsion movements was, however, the predominant feature.

Oppenheim further emphasized the association of hypotonia with hypertonia in these cases. The tendon reflexes often could be elicited only by reinforcement, and other evidences of muscular relaxation and flaccidity were present. This conjunction of hypotonus and hypertonus, the occurrence of clonic and tremor move-

3. Oppenheim: Ueber eine eigenartige Krampf Krankheit des Kindlichen und jugendlichen Alters; Dysbasia Lordotica Progressiva, Dystonia Musculorum Deformans, Neurol. Centralbl., 1911, xxx, 1090.

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ments, and his view as to the organic nature of the affection represent the chief differences from the original description of Ziehen. He further advanced the theory that the disease is caused by an anatomic lesion in some portion of the central nervous system which controls muscle tonus, bearing in this relation a certain analogy to paralysis agitans and the double athetosis. He suggested "dysbasia lordotica progressiva" or "dystonia musculorum deformans" as better descriptive names for the disease.

In the same year Flatau and Sterling[4] reported similar cases and confirmed the clinical picture as outlined by Ziehen and Oppenheim. In their cases, the early age insidious onset, gradual progression, the racial tendency and general character of the muscular spasm were all present. As in the original descriptions, the muscles of the face and articulation were not affected, and the torsion spasm was confined to the extremities and trunk. The movements were involuntary, tonic and twisting in character, often producing curious spiral movements of the trunk. They were not rhythmic or harmoniously bending, however, as in athetosis, and a certain monotony and stereotypy were quite evident. In their cases certain of the spasmodic movements were relieved by slight accessory movements in other parts (*Hilfsbewegungen*), suggesting the method of relief occasionally encountered in tic convulsive. Sometimes the movements were quick, like those of chorea, and occasionally were of such amplitude as to suggest the hemiballismus of Kussmaul. Occasional tremor movements suggestive of paralysis agitans were also observed. There were no true associated movements, but frequently irregular and bizarre contractions in other muscle groups accompanied a voluntary act. The tendon reflexes were not exaggerated, but were diminished or absent, and there was no hypotonia.

4. Flatau and Sterling: Progressiver Torsions Spasmus bei Kindern, Ztschr. f. d. ges. Neurol. u. Psychiat., 1911, vii, 586.

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Like Oppenheim, they regard the affection as organic, and emphasize its close relationship to the double athetosis. It differs, however, in localization, the trunk and proximal segments of the extremities being chiefly affected and in the type of the movements, namely, the twisting, torsion spasm with sudden brusque, clonic movements, and occasional tremors. They would call the affection "progressive torsion spasm."

In June, 1912, this subject was presented by Fraenkel[5] to the New York Neurological Society, and four cases of the disease were recorded, two of which have at various times been under my observation, and one I presented to the New York Neurological Society, in 1908, as an unusual case of myoclonia.[6]

Fraenkel's study verified the accuracy of the earlier clinical observations, and like Ziehen he relegated the affection to the degenerative neuroses, grouping it with tics and spasmophilias. He suggested "tortipelvis" as a distinctive designation for the affection. An excised fragment of muscular tissue from one of Fraenkel's cases was examined by Harlow Brooks and showed possibly some slight parenchymatous, but no atropic changes. In two of his cases intraspinal injections of magnesium sulphate were given with only temporary abatement of the spasm.

Since these publications, a number of observations have been recorded both in this country and abroad, showing that the disease is by no means a rare one. In this country Spiller,[7] Climenko,[8] Beling,[9] Abrahamson,[10] and Diller[11] have all reported cases which

5. Fraenkel: Dystonia Musculorum Deformans-Tortipelvis, Jour. Nerv. and Ment. Dis., 1912, xxxix, 361.
6. Hunt, J. Ramsay: A Case of Myoclonia of the Trunk Muscles, Jour. Nerv. and Ment. Dis., 1908, xxxv, 656.
7. Spiller: Case of Dystonia Musculorum Deformans, Jour. Nerv. and Ment. Dis., 1913, xl, 529.
8. Climenko: Case of Dystonia Musculorum Progressiva, Jour. Nerv. and Ment. Dis., 1915, xlii, 167; Med. Rec., New York, 1914, lxxxvi, 1000.
9. Beling: Case of Dystonia Musculorum Progressiva, Jour. Nerv. and Ment. Dis., 1912, xxxix, 461.
10. Abrahamson: Case of Dystonia Musculorum Deformans, Jour. Nerv. and Ment. Dis., 1913, xi, 38.
11. Diller: Dystonia Musculorum Deformans, with Report of a Case, Jour. Nerv. and Ment. Dis., 1916, xliii, 337.

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conform very closely to the original clinical picture as drawn by Ziehen, Oppenheim, Flatau and Sterling.

Spiller's case was somewhat unusual in that the patient was not of Jewish blood; two sisters were also afflicted with the disease and all were mentally defective.

I may mention that Bregmann,[12] like Flatau and Sterling, failed to find the hypotonicity on which Oppenheim had laid so much stress; he also observed a myotonic reaction in the muscles of the forearm, and at times the spasm was quite painful.

In a very interesting case described by St. Bernstein,[13] the duration of the disease was twenty-five years, and for a number of years the affection had been stationary, having apparently run its course. In this case there was distinct dysarthria, which has not been noted by any other observer. This disturbance of the speech St. Bernstein believed to be a dystonic phenomenon, dependent on hypertonicity and spasm of the muscles of articulation.

As thus far the muscles supplied by the cranial nerves have not been affected in this disease, I think we should accept this observation with some caution and consider the possibility of high involvement of the neck muscles supplied by the upper spinal nerves and its possible secondary effect on articulation.

To summarize, the progressive torsion spasm is a disease of childhood first described by Ziehen, and with rare exceptions has been confined to descendants of Russian and Polish Jews. It is essentially a progressive torsion spasm of the trunk and extremities, often associated with hypotonia and sometimes with movements of a more clonic or rhythmic character, suggestive of chorea and athetosis. Tremor movements are occasionally noted. The characteristic feature is the twisting and tractile quality of the spasm with the attendant distortion of the trunk and extrem-

12. Bregmann: Zur Kenntniss der Krampfzustände des jugendlichen Alters, Neurol. Centralbl., 1912, xxxi, 885.
13. St. Bernstein: Ein Fall von Torsions Krampf, Wien. klin. Wchnschr., 1912, xxv, 1567.

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ities, and the bizarre disturbances of posture, gait and station. Sensation is intact, and there are no symptoms of pyramidal tract involvement. It is a progressive affection with a limited symptomatology, and may eventually become stationary.

ANALYSIS OF PERSONAL OBSERVATIONS

Symptomatology and Types.—Six cases of this curious disease have come under my personal observation, all of the patients being at various times in the neurologic wards of the Montefiore Home and Hospital, which affords an unusual opportunity for prolonged and systematic observation.

In all the clinical picture corresponded very closely to the descriptions of Ziehen, Oppenheim and Flatau. In all of the cases save one, the affection began in childhood (6, 8, 9, 10 and 11 years, respectively), the exception being a typical case in which the first symptoms appeared at the age of 17 years. The sexes were equally divided, and all the patients, with one exception, were of Jewish origin.

The affection uniformly made its first appearance in one of the lower extremities, in four of the cases an interval of from one to two years elapsing before the opposite limb was involved.

In one case, the distribution in the extremities was hemilateral for a period of nine years (Figs. 1 and 2). In no case was the hand or arm the seat of primary involvement, although the upper extremities were affected in the later stages in three cases.

The lower portion of the trunk and the lower extremities were always most constantly and most severely affected, and a slow but definite progression was noted in all of the cases without exception. The muscles supplied by the cranial nerves were not affected, namely, those subserving the functions of articulation, facial expression and the ocular excursions. In one of the severe cases with high involvement of the neck muscles, there occurred, during an

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access of the motor paroxysm, slight contractions of the lower portion of the face, which I regarded as purely secondary, the result of a great effort to counteract the spasms.

In one of the cases the motor disability was so severe and so distinctly limited to the lower extremities as to suggest a paraplegic or pseudoparaplegic type (Figs. 3 and 4).

The character of the motor disturbance varied somewhat in the different cases, in this harmonizing with the reports of other observers. The tonic or hypertonic form with slow, torsion, twisting spasms of large muscle groups of the trunk and extremities predominated and was observed in all of the cases. Deforming functional contractures accompanied this type, which could always be overcome by strong passive movements. Especially characteristic in some of the more advanced cases has been a plantar flexion of the foot with extreme flexion of the toes producing a marked incurvation of the plantar surface (the semilunar foot, Fig. 5).

There was also a more active stage, characterized by movements of clonic or rhythmic character, accompanied by great motor agitation and unrest, suggesting somewhat the restlessness of chorea or athetosis. Two of the cases belonged to this type and produced the clownish movements, the bizarre postures and progression which suggested to Oppenheim such descriptive terms as the "dromedary gait" and the "monkey gait." In these cases the face was flushed, there was sweating and tachycardia, and the general motor excitement was at times so extreme as to be most distressing and alarming, requiring at certain periods morphin and scopolamin for relief. In two patients running was better performed than walking, and retrograde progression was also noted. In the other patients, the spasm was more tonic and much less active in character, and in bed or in a chair they rested with considerable comfort. In the late stage of this

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more tonic form a pseudoparalytic condition supervened, which was not paralysis in the ordinary sense, for some power of movement was retained, but paralytic so far as any voluntary control of the extremities was concerned.

Definite hypotonia was present in four of my cases, and in two it was absent, so that I do not regard this as a constant symptom in all stages of the disease. The tendon reflexes were present in all, although usually elicited with difficulty, and often only after repeated attempts, or during some favorable moment of relaxation. Hypertrophy of certain muscle groups was occasionally observed, as well as slight general wasting in the late stage, but without degenerative electrical changes.

None of the characteristic phenomena of myotonia were demonstrable in any of my cases.

There was no intelligence defect, and all of the patients were of average mental development. There were no hysterical manifestations or stigmata, and in none of the cases was there any tendency to stammering, impulsive speech, fixed ideas or phobias, which are so common in the degenerative motor neuroses. Nor were there noted any of those slight movements (*Hilfsbewegungen*) for the relief of spasm, so often present in cases of torticollis, such as laying the forefinger on the chin.

All of my patients would assume a definite fixed position while sitting or recumbent, which gave them most relief from the spasm. Occasionally, certain voluntary movements of an arm or leg would be made to influence or alleviate the severe spasm, experience showing that such movements had a relaxing effect on the dystonic disturbance. This was not, however, a psychic mechanism, and similar manifestations are occasionally observed in athetosis.

Remissions and Exacerbations.—Generally speaking, the disease was always gradually progressive. Occasionally, a slight improvement or remission was noted

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in some of the cases, but this was always of comparatively short duration.

A rather sudden progression occurred in one case while under observation (Fig. 6). This was an advanced type with involvement of all four extremities and the trunk. The spasm made its appearance rather suddenly in the neck muscles, which had up to that time been free. This nuchal spasm grew rapidly worse, and the attempt to control it seemed to disorganize the entire motor stability of the patient, and there ensued a period of about six weeks during which the child rolled and tossed in a veritable agony of movement. No position was found that would give relief, and I recall few more distressing spectacles than that of this child with a perfectly clear mind, struggling days and nights in the grip and conflict of these massive involuntary movements. During the few hours of sleep which came on after exhaustion, there was complete repose, but on regaining consciousness the spasms and torsions returned with unabated violence. Morphin and scopolamin were given to alleviate the suffering, and it really looked for a time as if fatal exhaustion would ensue, but the spasms finally grew less violent, it became possible to find a position which gave relief, and eventually the former conditions were resumed — chair-ridden and in certain postures fairly calm, with only occasional dystonic or torsion spasms. The control of the neck muscles was left permanently impaired, however, so that now no movement of the head is possible without strain and consecutive spasm (Fig. 7).

It would seem, in an exacerbation of this kind, as if the further impairment of what remained of this controlling mechanism jeopardized the compensatory stability which had been acquired during the gradual progress of the malady.

The Character of the Movements and the Nature of Torsion Spasm.—The movements varied in quality and character in the different cases and at different

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stages of the disease. There was always spasm, occasionally tremor, and sometimes movements of a choreiform or athetoid type.

In the early stage when an extremity is first affected, there is a disturbance in the harmony of movement; the foot turns outward or inward in walking, or the leg is thrown abruptly outward. In one case, a fine tremor movement always preceded and accompanied the spasm. Later, the spasm becomes more and more prominent and assumes the characteristic features of twisting and torsion, a variable hypertonia or dystonia in which the various muscle groups contract and relax. When the disease is well advanced, it is my experience that during rest those muscle groups showing spasm are more or less constantly in spasm, while their antagonists are usually relaxed and hypotonic.

The torsion spasm shows, therefore, a certain monotony or stereotypy after it is well advanced, and at rest the only signs may be repeated dorsal flexion of an ankle with an occasional spasmodic flexion movement of the knee or hip.

Any active movement, however, especially the effort to stand or walk, brings on a much wider dissemination of the spasm. The limbs are then drawn into weird postures or thrown out at strange angles, and walking or standing is quite impossible without assistance (Figs. 6 and 7). These are not true associated movements, but are associated dystonic phenomena.

In this stage the great involvement of the trunk muscles and hip girdle is quite apparent, producing lordosis with prominence of the buttocks and lateral distortion of the trunk from spasm of the erector spinal group and abdominal muscles. When the patient attempts to stand in this stage of the disease, there is not the normal postural fixation of the muscles. Instead, the muscles pursue quite independently their respective hypertonic and hypotonic tendencies, and the involuntary nature of the dystonia becomes very evident.

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There is in this late stage not only an absence of postural reactions of the normal tonus in standing, but also definite disturbance of the voluntary control of tonus in the affected muscle groups. For example, if the effort is made to carry out a plantar flexion of the foot when the foot is in dorsal spasm, the only response is a further increase of the extensor spasm, the exact reverse of what the voluntary effort is attempting to accomplish. The command is understood, the effort is made, but the antagonist which is in a state of hypertonia receives the impulse and not the agonists. This effort may be repeated over and over again with the same result. If, however, the request is made for a dorsal flexion of the foot, which is already in a state of strong dorsal flexion, instead of there being an increase of the already existing dorsal flexion, there is a sudden movement in a reverse direction, that is, a plantar flexion. In other words, there is an inability to dissociate harmoniously the essential tonus elements of a simple movement, namely, contraction of the agonists and decontraction or relaxation of the antagonists. A similar reversal of normal voluntary movement may also be observed when the foot is in strong plantar flexion, that is, an attempt to extend the foot only increases the already existing plantar flexion, whereas a voluntary effort in the direction of the already existing flexion produces a reverse movement in the opposite direction. This phenomenon I would designate the paradoxical or reverse phenomenon of dystonia, and would emphasize its diagnostic importance in the differentiation from spastic, hysterical and other forms of spasmodic contractures. It is, I believe, a pathognomonic sign, but thus far I have observed it only in advanced cases in the pseudoparalytic stage. It was typically present only in the ankle joint, and in one case in the wrist joint as well.

The difficulty in motion is therefore not only that of overcoming spasm; this we see in spasticity and the rigidity of paralysis agitans; but also a disharmony

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between the contraction and relaxation of agonistic and antagonistic groups of muscles. Some observers, notably Lewandowsky,[14] regard the essential element in athetosis as an inability to dissociate movements, so that any single voluntary effort is split up into numerous associated movements. In contrast, I would regard the essential disturbance in torsion spasm or dystonia as an inability to dissociate the tonus elements of innervation, which are the underlying factors in every movement. There is in addition the absence of that inhibition or control which is a characteristic of all involuntary movements, such as tremor, chorea and athetosis, and which is an especially important factor in the regulation of the tonus mechanism.

Unusual Clinical Types.—In my series of cases, there were two which presented somewhat unusual features and might simulate other forms of organic disease of the central nervous system. For this reason the clinical histories will be given in more detail.

CASE 1.—*Hemiplegic type of progressive torsion spasm* (Figs. 1 and 2).—A girl, aged 20 years, a descendant of Polish Jews, gives a negative family history, and no similar affection has occurred in any of the other members. The circumstances of her birth were normal, and the early history presents nothing of note except a tardiness in learning to walk and talk. The menses appeared at the age of 14 years and have been regular and in all respects normal. She had measles in early childhood and diphtheria at the age of 13.

Onset.—The first symptom of the torsion spasm appeared in her tenth year and caused a "limping" and awkwardness in the movements of the right leg. The foot was often thrown outward in a curious aimless sort of way and the muscles, especially of the foot, were stiff and contracted. She was taken to an orthopedic clinic and a cast was applied for three months, but after its removal the condition had not improved and grew gradually worse. There was no pain, only spasm and the tendency to awkward movements while walking.

Two years later the right hand showed symptoms of the disease, the earliest effect of which was noted in the handwriting. The fingers and muscles of the hand would stiffen

14. Lewandowsky: Ueber die Athetose doublé, Deutsch. Ztschr. f. Nervenh., 1905, xxix.

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and cramp very much as in writer's cramp, and if the effort to write was persisted in, the hand and forearm would be forcibly extended or thrown backward. This difficulty with writing came on insidiously and progressed gradually, and at last she gave up entirely the use of the right hand and learned to write with the left.

For nine years the torsion spasm was limited to the extremities on the right side, gradually growing worse, and during this time there was not the slightest involvement of the left arm or leg. At the age of 19 she first noted a tendency to slight torsion in the left hand and arm in writing, and during the past few weeks a subjective sense of spasm in the left leg. (She states that she is aware of the impending involvement of an extremity by a drawing sensation which precedes any actual objective manifestation.)

The face, speech, deglutition, and ocular and neck muscles are not affected.

Examination (December, 1915, at the age of 19).—She is very short in stature with well developed muscles. The pupils are equal and react promptly to light and accommodation. There is no nystagmus. The cranial nerves are all negative. There are no tremors, and the sensations, superficial and deep, are quite normal.

Motility: When the right arm is held out from the body the hand is slightly flexed at the wrist, and the fingers extended somewhat suggestive of an athetoid posture. There are slight spasmodic variations in the position of the fingers. The general musculature of the arm during test is firm and generally spasmodic. Occasionally, there will be a sudden change in the position like a flexion of the wrist or elbow, but the arm immediately is restored to its former position. There are no associated movements, nor does the spasm seem to be increased by movement of the other extremity.

There are no evidences of motor disturbances in the left arm, during the ordinary tests for motility. During the act of writing, however, the fingers stiffen and assume a cramped attitude as in writer's cramp, and there is very evident stiffening of the arm muscle groups, which is apparent even in the deltoid and scapular muscles. The scapula is occasionally drawn out from its natural position. The masseter reflex is active, and the supinator, biceps and triceps jerks of the left arm are active. The tendon reflexes of the right arm are masked by continual spasms; occasionally, however, in moments of relaxation, the responses are elicitable. The myotatic irritability is somewhat increased. Respirations are normal and there is no cramping or stiffening of the abdominal muscles in the recumbent position. Abdominal reflexes are present and active.

Lower Extremities in Recumbent Posture: The right is held slightly flexed at the knee and everted. There is little or no spontaneous movement at rest, except occasionally a

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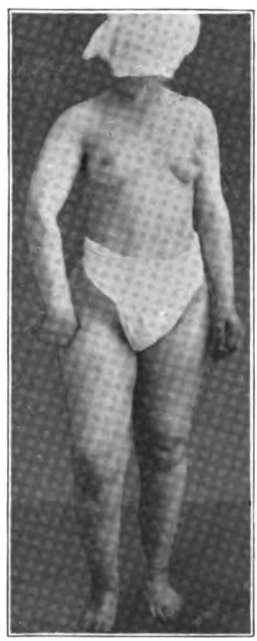

Fig. 1.—Hemiplegic type of progressive torsion spasm showing the attitude in a case of hemidystonia dextra of eight years' duration; subsequently, the left arm and leg showed evidences of involvement.

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Fig. 2.—Posterior view, hemiplegic type of progressive torsion spasm; hemidystonia of eight years' duration.

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Fig. 3.—Paraplegic type of progressive torsion spasm of six years' duration; the symptoms of dystonia are limited to the lower extremities and lower portion of the trunk.

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Fig. 4.—Posterior view, paraplegic type of progressive torsion spasm; dystonia paraplegica.

Fig. 5.—Paraplegic type of progressive torsion spasm (recumbent posture). Note the general wasting of the calf muscles, and the semilunar foot.

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Fig. 6.—Advanced case of progressive torsion spasm; involvement of the trunk, neck muscles and all four extremities. Note the bizarre, clownish attitude and the poorly developed musculature of the lower extremities.

Fig. 7.—Advanced case of progressive torsion spasm in the recumbent posture of election (see Fig. 6).

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slight flexion of the toes. Any attempt to move the leg immediately produces a slow torsion spasm and twisting of the whole extremity. The toes are cramped, and the muscles of the leg and thigh become rigid and show slow, twisting movements. None of these objective symptoms are present in the left leg. (Subjectively, however, for the past few weeks there has been a sense of uneasiness and nervousness in the left leg and a tendency for the left leg to draw up.) The erector spinae group on the right side are stiff and in spasm; on the left they are relaxed. There are certain postures both while sitting and recumbent which afford her a certain relief, but there is no method by which the spasms may be controlled, other than by posture. The knee jerk and the Achilles jerk on the right side vary with the degree of spasm, but in moments of relaxation they are quite active. There is no hypotonia. The plantar reflex is normal. Tendon reflexes on the left side are present and active.

Standing: She stands on the left leg with the right leg slightly flexed at the knee. There are occasional movements of toes or knee, and the muscles of the right leg are firm and in spasm. The spine is fairly straight with a slight tendency to lumbar lordosis, giving the appearance of an undue prominence of the posterior pelvic and gluteal region. The attitude is definitely hemiplegic, except that the right hand is thrown back posteriorly, with the little finger abducted in a bizarre attitude. The gait is of a somewhat hemiplegic character, but is unlike a spastic gait because of the curious motor spasm which takes place in the midst of a step, changing and distorting the position of the limb.

In sleep she assumes certain exceptional positions which produce relaxation and relief; once asleep she is not disturbed and all spontaneous movements cease.

The spasms cause no pain, but produce a stiffening sensation and a distinct sense of fatigue in the right arm and leg. Spasms are diminished in the recumbent posture, increased on standing and walking and therefore intensified on motion.

The electrical examination of the muscles shows no deviation from the normal. Heart, lungs and abdominal viscera are negative. The urine is normal. The Wassermann reaction is negative.

Intelligence is normal.

Comment: The torsion spasm in this case is interesting because of the hemilateral localization over a period of nine years (hemiplegic type). The condition, however, is steadily progressive and slight, but definite involvement of the left side is now evident.

The spasm is much more of the tonic torsion type, and there are no tremors, no choreiform movements and only the slightest suggestion of an athetoid tendency in the fingers of the right hand.

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The case has at no time during the past year shown any of the agitated tendencies sometimes observed in these cases.

CASE 2.—*Paraplegic type of progressive torsion spasm* (Figs. 3, 4 and 5).—A girl, aged 12 years, of Jewish parentage; gives no family history of any similar disease. The birth was normal and she was breast fed until the fifteenth month. Her early development was normal. She had measles at the age of 3.

Onset.—The first symptoms of torsion spasm appeared at the age of 6 with a dragging of the right foot and a tendency to throw the right leg outward in an aimless manner while walking. A plaster-of-Paris cast was applied for three months without improvement. During the course of the next few months the muscles of the right foot and leg became more drawn and contractured. One year later the left leg was similarly involved. These symptoms have gradually progressed, and since the age of 9 she has been unable to stand or walk. Numerous tenotomies were performed without material benefit.

The face, speech, arms and trunk muscles are not affected.

At this time (ninth year) the child was brought to the Vanderbilt clinic for examination. The lower extremities were then in a state of tonic spasm; the legs were extended; The feet and toes in plantar flexion. Some of the muscle groups of the legs showed momentary hypotonia. The trunk was not affected. The tendon reflexes were elicitable with difficulty. There was no clonus, and the plantar reflex was difficult of interpretation because of the marked plantar spasm of the foot and toes. The Babinski phenomenon, however, was not present. The muscles were in a state of hypertonicity, and there was only moderate torsion spasm. No associated movements were noted. The movements of the arms were normal, and the supinator, biceps and triceps jerks were normal. The abdominal reflexes were active. The movements of the face, mastication, articulation and the ocular excursions were quite normal. The pupils were equal with prompt responses. Intelligence was normal.

This case at this time was regarded as one of dystonia musculorum deformans of unusual type, although the resemblance to a spastic paraplegia with contractures was rather striking. Subsequently, a bilateral subtemporal decompression was performed at the Polyclinic Hospital, presumably for the relief of a supposed cerebral lesion. The child made a good recovery from this operation, which naturally was without effect on the subsequent course of the disease.

Two years later she was admitted to the neurologic wards of the Montefiore Home and Hospital.

Since the previous examination there has been a distinct progression of the spasm upward, with involvement of the hip girdle and lower portion of the back and abdomen. In

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the recumbent posture there is marked torsion spasm of the muscles of the erector spinae group in the dorsolumbar region on the left side. The corresponding muscle mass on the right side is somewhat relaxed, and there is a constant massive torsion and twisting with variable scoliosis and kyphoscoliosis. The lower portion of the abdominal wall also participates in the spasm.

The right lower extremity is extended and the foot is held in a position of extreme plantar flexion including the toes, producing a conspicuous concavity of the plantar surface (the semilunar foot [Fig. 5]). Some of the muscle groups are hypotonic and others hypertonic and in spasm, which fluctuates in intensity from time to time. Occasionally, the leg will be drawn up in sudden flexion.

The position of the left lower extremity is changed from time to time by slow constant flexion movements of the knee and hip. As on the right side there is an exquisite semilunar deformity of the foot with plantar spasm of the toes.

With the fluctuations in the hypertonicity, a hypotonus of certain muscle groups is quite evident on palpation. Active or passive movements produce a considerable increase in the tonic phenomenon with spasms and distortion of the extremities. There are no tremors, athetoid or choreiform movements, and no true associated movements. Any of the contractures may be overcome by firm traction and pressure, and there is no structural fixation of the joints.

There is some, but very slight power of voluntary control of extremities, so great is the degree of the dystonic disturbance. In the distribution of the involuntary spasms there is a certain monotony and stereotypy, so that it varies in localization but little from day to day. The child is fairly comfortable in the recumbent posture of election, but any change from this position increases the tendency to involuntary spasm and strain. In attempting voluntary movements of the ankle the paradoxical or reverse phenomenon of dystonia is very evident; for example, if the foot is in a state of tonic dorsal extension and the command is given to flex the foot, the result is an increase of the extension spasm. If the command is now given to extend the foot, that is, a voluntary effort in the direction of the involuntary spasm, the reverse movement takes place and vice versa if the foot is in plantar spasm. The same phenomenon may sometimes be demonstrated in movements of the knee, but is especially characteristic and well marked in the ankle.

An attempt to carry out a certain definite movement of an extremity is accompanied by tonus disturbances in other groups of muscles of that extremity, and even on the opposite side.

The child is unable to stand alone, but if supported on either side, the muscles of the legs and hip girdle fail to respond to the postural reflexes, and the variable spasm and

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relaxation goes on involuntarily and in a perfectly aimless manner. The legs will be suddenly drawn up, or there is performed a series of slow involuntary scratching-like movements with the foot, but there is not the slightest trace of any coordinated effort on the part of the legs to stand or support the trunk. In this position the spinal column is perfectly free and mobile, but its lower portion tends to be drawn by spontaneous contraction into kyphoscoliosis.

The knee jerks are elicited, but with difficulty, and this is especially true of the Achilles jerks. The plantar reflexes cannot be tested very satisfactorily owing to the marked plantar spasm. There is no Babinski response. The abdominal reflexes are active. The arms, upper portion of the trunk, neck and all muscles supplied by the cranial nerves are normal in every particular. The sensations are normal. The musculature of the lower extremities below the knees is generally atrophic, but this is a secondary atrophy and there are no qualitative changes in the electrical reactions. The pupils are equal and react. The mind is perfectly clear, and intelligence is of normal standard. The optic disks are normal.

The urine, Wassermann test of the blood and the visceral examinations are all negative.

Comment.—This case is of unusual interest because of the paraplegic localization of the dystonic disturbance, the early appearance of the paralytic stage, and during the earlier period the moderate degree of involuntary torsion movements, thus simulating a spastic paraplegic state.

NATURE OF THE DISEASE

In agreement with Oppenheim and most subsequent observers, I regard the progressive torsion spasm as an organic affection of the central nervous system. In favor of this view may be mentioned the gradual progression, the peculiar characteristics of the motor disturbance, its association with tremor, choreiform and athetoid movements, the gradual generalization of the spasm until the entire voluntary musculature is involved, sparing only the muscles supplied by the cranial nerves, and finally the absolute chronicity of the clinical picture.

There is also important negative evidence in the absence of hysterical stigmata and the mental characteristics of the convulsive tic, which usually play so important a rôle in the functional motor neuroses of hysterical or psychogenic origin.

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Any attempt to localize the lesion in the present state of our knowledge is purely a matter of conjecture. It is, however, not without interest to theorize in this particular, so that when an opportunity is offered for pathologic investigation, the various theories which have been offered may be confirmed or refuted.

In a recent pathologic study of the juvenile paralysis agitans, I found as the essential lesion an atrophy of the motor cells of the globus pallidus system,[15] that is, not only the large multipolar cells of the globus pallidus proper, but also the large multipolar cells of similar form which are scattered among the smaller ganglion cells of the putamen and caudate nucleus. I therefore regard the juvenile paralysis agitans as a true system disease limited to the efferent motor cells of the globus pallidus mechanism, and consider this to be the essential lesion underlying the paralysis agitans syndrome. In other words, this motor mechanism of the corpus striatum plays an important rôle in the control of the extra-pyramidal motor system, through its connection with the red nucleus, the corpus subthalamicum and substantia nigra, an essential element of which is the regulation of tonus. In the corpus striatum of Huntington's chorea, these large motor cells are preserved in both the putamen and caudate nucleus amid the extensive atrophy and disappearance of the smaller ganglion cells. These smaller cells apparently exercise a coordinating and inhibiting influence on the large motor cells of the globus pallidus system, and when this inhibitory function is lost, there results chorea of the Huntington type. The Vogt[16] syndrome and Wilson's[17] syndrome of the corpus striatum representing, according to my conception, different degrees of involvement of these two cellular systems, the one motor the other coordinating and inhibitory.

15. Hunt, J. Ramsay: Progressive Atrophy of the Globus Pallidus, Tr. Amer. Neurol. Assn., May, 1916.
16. Vogt, Cecile: Syndrome du corps strié, Jour. f. Psychol. u. Neurol., 1911, xviii, 479.
17. Wilson: Progressive Lenticular Degeneration, Brain, 1912, xxxiv, 295.

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I would therefore regard the corpus striatum as a subordinate motor mechanism, having as its chief function the control of automatic and associated movements. It is composed of two cellular systems—the large cells of the globus pallidus which represent the motor nucleus proper, while the smaller cells of the putamen and caudate represent a cortical mechanism which exercises a controlling and inhibitory function.

This interpretation of the functions of the corpus striatum is not without interest to the subject under discussion, for many of its symptoms are also encountered in dystonia.

All writers on the subject of the progressive torsion spasm emphasize its close relationship to athetosis and the frequent occurrence of tremor and choreiform movements, so that in any future pathologic investigations the cells and fiber systems of the corpus striatum should receive careful study.

My own conviction is that a lower type of mechanism is at fault, one which is closely associated with the corpus striatum, but which is engaged in the regulation of tonus, especially in its relation to the reciprocal activities of agonistic and antagonistic muscles, for this I believe constitutes one of the essential factors in the motor disturbances of torsion spasm. The preservation of facial expression and articulation is also against a localization in the corpus striatum, for we know from many pathologic studies that the face and articulation are both involved in this localization.

CONCLUSIONS

As a result of this clinical study, the following conclusions may be suggested:

The progressive torsion spasm of childhood (dystonia musculorum progressiva) is a progressive organic affection of the central nervous system.

There is the gradual loss of a mechanism which is engaged in regulating and controlling tonus during the voluntary, automatic and reflex activities of muscles. As a result, there is a disturbance or loss of

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those reciprocal tonus activities of agonistic and antagonistic muscles which are a part of every complex or synergic movement. This, together with the loss of inhibition, I regard as the chief factors in the production of the motor disorder.

It is a definite disease, and, like such affections as paralysis agitans, double athetosis and dyssynergia cerebellaris progressiva,[18] runs a progressive course and then remains stationary, other portions of the nervous system remaining unaffected.

It is closely related to athetosis and yet in its typical form is readily differentiated from this affection. Transition or combined forms probably occur.

It is essentially a disease of childhood, but has appeared as late as the seventeenth year.

The face and muscles of articulation are not affected except in the terminal stage, when there is involvement of the muscles of the neck. Such mild disturbances as have then been noted are probably of secondary origin.

With the characteristic torsion spasm there is usually but not always, associated a not inconsiderable degree of hypotonia.

There may be a hemilateral involvement of the extremities which may exist as such for some years. A pseudoparaplegic or paraplegic type may also occur.

The torsion movements continue, but are diminished during rest, and are greatly increased by any muscular activity, especially standing or walking; they cease entirely during sleep. Other disorders of motility, as tremor, chorea or athetosis, are frequently associated.

The trunk and lower extremities are chiefly affected, especially their proximal segments.

In the later stages there is often a well marked tendency to stereotypy or fixation in the distribution of the spasm, which predominates in certain muscle

18. In addition to the references already given, the following will be found of interest:
Bonhoeffer: Torsions Spasmus, Neurol. Centralbl., 1913, xxxii, 137.
Beach: Torsions Krampf, Neurol. Centralbl., 1915, xxxiv, 654.

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groups. In this stage, I have observed in the movements of the ankle, and less commonly of the wrist, a curious reversal of the normal manifestations of tonus which may be termed the paradoxical or reverse phenomenon of dystonia. When present it is, I believe, a pathognomonic symptom.

The affection varies considerably in the degree of motor unrest and agitation which is produced. This may occasionally assume sudden and violent proportions requiring strong sedatives and reducing the patient to a dangerous state of exhaustion. It may occasionally constitute an agitated form or stage of the disease.

The affection is progressive and incurable and, with few exceptions, has appeared only in descendants of Russian and Polish Jews.

20 West Fiftieth Street.

ABSTRACT OF DISCUSSION

DR. HUGH T. PATRICK, Chicago: This is a rare opportunity to see a typical example of this interesting malady. But first it might be of some interest and, perhaps, of some value, to state very briefly the error I made on this case about three years ago. This boy, by the way, is not of Polish-Jewish origin. The parents are Hebrews, but both father and mother say that so far as they know none of their antecedents have been of the Russian-Jewish folk. When I first saw this boy in September, 1913, the condition had been progressing for about five months. He was then 7 years of age and the family and personal histories were absolutely negative. His symptoms began with a tonic contraction of the right leg. Then a little later there was added to this tonic contraction occasional jerks which we might call clonic spasm, but which was simply a jerk of the leg. The diagnosis I made was hysteric monoplegia, for the following reasons: The boy held the right leg semi- or partly flexed at the knee and at the hip. He walked in a peculiar way which was strongly indicative of functional disease. Before Ziehen presented this subject, an assistant of his had shown the cases and thought the condition was hysteric. Oppenheim was also inclined to make a diagnosis of hysteria in his first cases. I do not say this to excuse myself, but to put on their guard those who may subsequently see these cases. This boy walked in a very peculiar way, and we know that most of the bizarre, non-typical gaits are very apt to be manifestations of hysteria. He would advance the

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affected leg, touch the toe to the floor and then hop with the left leg. When I requested the boy to extend the right leg he apparently tried to do so, and then there were these intermittent jerks in the leg and he was unable to extend it. In the act of turning a summersault the affected leg straightened at the knee in a perfectly normal way.

I believe that at that time the left leg was already beginning to show involvement. When I began to examine it there occurred a slight tonic extension and when requested to move the foot at the ankle the boy was unable to do so. This I also attributed to functional disease. In other respects results of examination were negative. The subsequent history was given me by Dr. Haass, but I ought to say that later the case was seen by Dr. Camp of Ann Arbor, who first made the proper diagnosis. Later a well-known Eastern neurologist considered it a case of Wilson's disease. The especial value in seeing one of these cases is to learn the peculiar type of movement. It is a combination, as Dr. Hunt has indicated, of various types of hyperkinesia. There is something of the choreic, there is something of the tics, there is something of the athetosic. One particular feature in these movements is a sudden, quick movement, even quicker than most of the tics, that occurs occasionally. Most of the movements are rather slow, but occasionally there is this sudden quick jerk, generally extensor. This is a symptom which is so disconnected from all other organic movements that it is particularly valuable in the interpretation of this suppositious dissociation, because there is something there certainly more than dissociation; it is either an excess of impulse, or an absence or a great diminution of inhibition.

Dr. E. W. Haass, Detroit: Dr. Patrick has given a brief preliminary history of this boy whom I saw a little over two years ago, he then having been ill something over a year. When I saw him first he was practically in this condition of helplessness. Diagnosis had already been made by Dr. Camp, who had been observing the patient for about six months before he was brought to me. Previous to this there had been a diagnosis of hysteria and for a short time a diagnosis of beginning pseudo-hypertrophic paralysis. A piece of the calf was taken for examination by Dr. Camp with a finding of simple atrophy. There is nothing in his family history. His eye reflexes are normal; ocular movements are normal; the deep reflexes, as far as elicited (it is very difficult to get these reflexes), are apparently normal; the superficial reflexes are all present. There are no sensory disturbances whatever; there are no electrical disturbances. He has had a diagnosis of Wilson's disease since Dr. Camp diagnosed the case. But his mentality has not been affected at all. Wilson's disease is a progressive lenticular deterioration plus mental deterioration plus a condition of biliary cirrhosis. The hepatic disturbances, however, have never been determined, except on

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post mortem. As far as we can ascertain the liver is normal in size and he responds entirely normally to the phenol-tetrachlor-phthalein test. This spring he was subjected to this test and his liver is able to excrete dye in an entirely normal manner. He is at rest during sleep. His motions are very much exaggerated during excitement, as here at the present time, and he has been in this condition of practical non-progression now for a year and a half. It began in his right leg and involves this extremity more than the left and, perhaps, his right arm more than his left. The duration of the case is now about three years and three or four months.

DR. A. L. SKOOG, Kansas City: Are there any restrictions to joint movements in the late stages.

DR. JAMES RAMSAY HUNT, New York: As to the diagnosis of this distressing condition, if one has seen a few cases it is generally pretty easy, certainly insofar as the differentiation from hysteria or the spasmodic tics is concerned. But even after I was fairly familiar with this disease, in the dystonia paraplegica I was uncertain as to the diagnosis for a considerable time. The localization in this case was limited to the lower extremities, the involuntary movements were very slight, and it was difficult to distinguish between the torsion spasm and a spastic condition, although there was no clonus and the Babinski reflex was not present. With the extension to the trunk and as the torsion quality of the movements became more evident, all diagnostic doubts were removed.

In none of my cases was there any permanent fixation of the joints. Strong passive movements were always sufficient to overcome any deformity resulting from the spasm. I would also lay great stress on the absence of involvement of the face and muscles of articulation, in excluding the double athetosis. The paradoxical phenomenon of dystonia, when present, I believe to be a pathognomonic symptom of this disease and an important differential sign in excluding other forms of muscular spasms. Thus far I have encountered it only in advanced cases in the paralytic stage, it is then especially characteristic in the ankle and sometimes the wrist joint. Apparently, the affection must reach an advanced stage before a complete reversal of the tonus innervation is possible. It is very significant that we have here a disease which affects principally the muscles of the trunk and extremities. It is not only an inability to dissociate tonus impulses to agonistic and antagonistic muscles, but there is also an absence of the normal inhibition or control. The disturbance apparently results from the loss of a mechanism which is engaged in regulating and distributing tonus impulses to the voluntary muscular system.

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THE FACTOR OF FEAR IN NERVOUS CASES

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In many cases of functional nervous disorder, the factor of fear is quite obvious. But in many cases, though equally important, it is not at once apparent. Of the latter, there are numerous varieties which may be divided into many groups. One group embraces patients known to have physical courage:

A few years ago there was referred to me the most noted pugilist of his day, a specimen of perfect physical manhood, known to be not only fearless in the ring but peculiarly carefree if not careless. He was suffering with what were considered rather vague and baffling nervous symptoms, principally insomnia, lack of interest, and moodiness. A careful analysis[1] soon revealed that some trifling symptoms due to high living and domestic friction had served to put the idea into his head that he was losing his mind. This phobia was his sickness, and the fear so possessed his soul that he was good for nothing until he got rid of it. Needless to say, the patient himself was quite unconscious of the nature of his trouble, and his physician had overlooked it.

A sheep rancher of Wyoming was referred to me because of insomnia, loss of appetite, abdominal distress, general nervousness and inability to look after his ranch. What really was the matter with him was fear of cancer of the stomach. This phobia completely unnerved him and caused him enormously to magnify every bodily sensation. But was he a nerveless coward? Decidedly not. There was a time when the cattlemen of the Far West made sheep raising a hazardous occupation. Through those dangerous years he lived without a tremor, though he never went to sleep without a rifle by his side. Once he was informed that three cattlemen had started out "to get him," and the information was correct. He mounted his horse and, properly armed, rode

1. This does not mean so-called psychanalysis. In this paper the tenets and methods of the freudian school are not taken into consideration.

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to meet them. As he expressed it, he "talked them out of it,"[2] and the three would-be assassins turned and rode away. In this encounter he was not in the least apprehensive or uncomfortable, and I learned of the incident only in casual conversation about his business.

A policeman, 49 years old, consulted me for intractable insomnia, head pressure, general nervousness and loss of weight. He was not a man to suspect of fear. For many years he had been on active service in one of the worst precincts of Chicago, and on account of his familiarity with criminals was frequently sent after the worst types. He had been in numerous "gun" fights. Once a notorious "gun" man standing beside him fired point blank at his head. All this disturbed his equanimity not a whit. And yet his illness was the result of fear, pure and simple. It came about in this way: a malicious person had preferred against him charges of misconduct, and he was cited to appear before the trial board. This worried him greatly. Innocent, he keenly felt the disgrace of the accusation and feared that he might be suspended or even discharged. He trembled for his well earned good name and for his home, on which there was a mortgage. Naturally, he began to sleep poorly, to have queer feelings in his head, and then to feel uncertain of himself. At this juncture some friends sympathetically told him that one could go insane from worry. These were the steps: fear of disgrace, fear of financial collapse, fear of insanity. But did the patient know all this? Not he. He knew only that he was nervous, that he suffered, and that he did not feel sure of himself.

A lovable old general, who had gone into the Civil War a private and served throughout the conflict with the greatest distinction, was sent to me because he was quite a nervous wreck. Twenty-five years before he had strained or sprained his back. The next morning he fainted and then rapidly developed what was called "spinal irritation." From this he suffered for many years, but finally made a relative recovery under the skilful guidance of Dr. S. Weir Mitchell. About four months before he came to me a saddle had turned with him, giving him a rather severe but (physically) not serious jar. At once all the old trouble returned. He was nervous and sleepless, and complained of pain and great weakness in the back. He never went out without a queer-headed cane which he could use as a support for the spinal column the moment he stopped walking and had to stand. The whole trouble was fear, vague as to object but very definite as to

2. His explanation of "talking them out of it" is not devoid of psychologic interest, and also shows how undisturbed he was. Essentially it was as follows: "Well, you see, the longer a man talks the less apt he is to shoot, and I got them to talking. And then, of course, they knew I was a crack shot and that if they made a move I'd be pretty sure to get one of them and maybe two before they could get me. And each of 'em kind of hated to be the first one."

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intensity. At first I did not tell him he was scared sick because he would not have believed me and I should have lost his confidence. But a course of training with the temporary use of bromid rather rapidly put him on his feet and made him comfortable; and he left for his home about twelve hours away. Unfortunately he took a road he never had traveled on, a rough one. The jarring not only disturbed sleep but started little "fear thoughts" about its possible effect on his spine. These worries kept him awake the next night, and in a panic he hastened back to Chicago. Even then he was so far from knowing that he was afraid that I had to explain to him the steps by which he had become the victim of fear, and clinched it by saying, "In short, General, you were a d—d coward." And this was true, although addressed to as brave a soldier as ever led his men. In other words, that a man is as brave as a lion is no reason why he may not have a paralyzing fear. He may be utterly devoid of fear of many dangers and sick of fear of one other.

A mine manager from Mexico came to be injected for trifacial neuralgia. And he made a big fuss every time I introduced the needle. As he said, "That little needle 'gets my goat'." At this time there were serious labor troubles in Colorado, and I happened to ask him if his labor was organized. He said it was not, but that once three labor organizers walked into his office and announced that they were going to unionize the mine. "What did you do?" I asked. "Kicked them out so quick they didn't know what had happened." "But," I said, "they might have shot you up." "Shoot up h—l," was the reply. "They didn't have time." That is to say, the revolvers of three aggressive labor leaders didn't "get his goat" at all.

This case is analogous to the phobias and, like the four preceding, illustrates a rule applying to many of them. We fear the unfamiliar; the things of which we are ignorant. All his life my patient had been used to tough men with pistols but not to physicians' instruments. German students are timid about bare knuckles but not a bit afraid of the Schläger that hacks their faces to pieces. The boldest financier may be inexpressibly scared about his liver. The one almost universal fear of the human race is Hades, or what follows death — the one thing of which we all know absolutely nothing.

But to this rule there are notable exceptions: exceptions which show what a fundamental and powerful

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emotion fear is, what a primitive race we are and how we still are largely controlled by instincts and emotions: not by knowledge and judgment.

A physician I never had seen hurried into my consultation room gasping for breath in a peculiar way. His mouth was open and he was taking quick, very deep inspirations at not too frequent intervals. Often he placed his hand over the precordium. It was easy to see that he was not in pain and had no organic dyspnea. He asked me to examine his heart. I asked him who had already examined it. He named half a dozen men more competent than I to pass on it. All had pronounced it normal. I declined. The man himself proved to be a competent and intelligent practitioner. A few years before he had run at top speed about half a mile to catch a train. He just caught it, clambering onto the rear platform as the train gathered headway. Of course he was out of breath and his heart was beating violently. At that moment came the thought: "What if I should have an acute dilatation now and here with no possible help?" The thought was terrifying, and he was in acute distress until he reached his destination, a short distance away. That was the beginning of a series of periods when the fear of heart failure would get the better of him, and in many of the attacks he breathed as when I saw him, the feeling being that the air did not go deep enough, did not reach the right spot.[3] There were times when every afternoon from 2 to 4, under one pretext or another, he would sit in a nearby drug store because he knew that then none of the physicians of the neighborhood had office hours; all were out making calls. And he just had to be where he could get quick assistance if necessary. If he were called to a house beyond the thickly settled districts he could not compel himself to cross the vacant spaces, but would turn about and go home, all because of the fear of sudden heart failure, his own knowledge and experience and the confident assurance of numerous heart experts to the contrary notwithstanding.

To state the thesis another way, intellectuality seems to be no effective protection against phobias. It is largely a question of temperament. The intellectual and cultivated are quite as susceptible as the stupid and ignorant.[4] And regarding this temperament one may frequently make a most interesting observation

3. This sensation is frequent in neuroses involving ideas of the heart, lungs and throat, and some others.

4. An intellectual and cultivated person may not be in the least "matter-of-fact." That he may be lacking in "common sense" is notorious.

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of what at first sight looks like a psychologic paradox. Concerning these phobia patients one very often hears members of the family say, "She never gives up," "Nothing discourages him," "He's a hard fighter," "She's full of courage and patience," and the like. All of which is perfectly true, and yet this person is in the clutches of an overpowering fear. The explanation lies in the nature of the individual. They are the people dominated by an idea. The absolutely undaunted young football player, who plays a whole period after he has been so battered that he is practically unconscious, does so because he is possessed by the idea that he's got to win and he's going to. The same boy may be just as much in the grip of an idea that he is ruined by masturbation. The mother who is absolutely tireless and never loses courage in the nursing of her child, because she feels that she must and will save it, may be just as strongly possessed by the fear that she may suddenly lose control of herself and cut the child's throat. The born enthusiast makes a fine phobic. Had her obsession taken another direction, Joan of Arc would have made a beautiful case of *folie du doute* or of agoraphobia. Martyrs and profound psychasthenics are often made of the same stuff.[5]

To insist that one should be careful not to confuse phobia with impulse, the fear of doing a thing with a sudden desire to do it, would seem to be a work of supererogation. But patients themselves frequently make this mistake, and physicians sometimes follow their example. One of the best English textbooks on insanity cites in some detail a typical case of phobias as one of homicidal impulse. Brief mention of a few cases will make the type clear:

A woman of 50 years habitually sat in a far corner of the room because she said she had an impulse to throw herself out of the window. I had the greatest possible difficulty in

5. I have recently seen a successful theological teacher and evangelist who was a missionary in darkest Africa where he heroically endured great hardships and undismayed faced manifold danger, yet now was suffering with an overwhelming fear of closed places.

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getting her to approach the window of my office (ninth floor) because of her overpowering fear that she might suddenly lose control of herself and jump out. She went toward the window haltingly with tense muscles and anxious countenance and clinging frantically to a heavy desk, as if some one were shoving her toward the window and she resisting.

A gentleman came to me in great distress of mind because he said he had an impulse to kill his two young sons. He had given away his revolver, had carried an ax about a mile away and thrown it into a river, and had disposed of the domestic clothesline in the same way, having had permanent wire put up in its place; all because the idea how easily he could kill the boys kept recurring with such intensity that he feared he might in some way suddenly do it.

A very devoted mother was in the same horribly distressing state of mind regarding her two little girls. She never went into the kitchen because the carving knives were there, had all meats carved in the kitchen, and persuaded her husband to take his razors out of the house—this because she was constantly in mortal terror for fear she might suddenly have an overpowering impulse to harm the children. What she came complaining of was nervousness and sleeplessness. Only after close questioning did she tearfully confess to an "impulse to kill the children."

A frequent fear, which the patient looks on as an impulse, is that of doing some mal apropos thing in public; of using obscene language or yelling or slapping someone or making a suggestive movement. Frequently such fears are really fear of insanity, of suddenly losing self-control. But the conception of the dreaded act recurs so constantly and forcibly that the patient gets the idea that he has a tendency to commit it, and the doctor takes his word for it. Needless to say, the diagnosis is vital for both prognosis and treatment, but is generally very easy. The patient at once acknowledges that he does not want to do the thing, never did want to do it, and has no reason for doing it, but fears that for some reason in some way he may suddenly have an abnormal impulse to do it, and will not be able to resist the impulse.

An interesting type of case, and one frequently misunderstood, is that in which the patient suffers from the physical results of the fear, but is quite unconscious that his trouble originated in fear and really

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still is a definite fear. As indicated before, these cases are very common. Sometimes it is easy to unearth the fear and its origin, sometimes difficult and occasionally impossible. An easy one was the following:

A young commercial traveler consulted me because of nervousness which came on in restaurants and hotel dining rooms. He dreaded to enter such a place because as soon as seated he became shaky and apprehensive, and had palpitation, precordial distress, soon followed by nausea and cold perspiration. For three months he had been unable to take a meal in a public eating place, which meant that he had been unable to work for that length of time. Only a few minutes were needed to learn that some months before, in a restaurant, he had been taken violently ill with nausea and profuse emesis followed by active diarrhea and marked prostration; probably ptomain poisoning. This most disagreeable if not alarming catastrophe was unconsciously associated in his mind not with something he had eaten but with his presence at table in a restaurant, and it was fear of a recurrence of the thing in a public place, surrounded by people and no doctor near, that caused his symptoms. Although the patient had had no suspicion that he was afraid, he recognized the logic of the situation when it was presented to him, and made a rapid recovery.

Not quite so simple was the following:

A gynecologist sent to me a young married woman with vaginismus. She had been married many months, but coitus had not been completed. No cause was obvious. The introitus was normal and not especially sensitive. The patient had no prudish notions regarding intercourse, was deeply in love with her husband and anxious to fulfil her functions as wife and prospective mother. Detailed inquiry seemed to show that the trouble was not so much local pain as instinctive resistance and overpowering inability to endure the act. I further found that not only could she not endure to have the husband touch the pudenda, but even his touching the breasts caused a very disagreeable sensation with a strong feeling of defense. The cause of this great and greatly regretted repugnance was totally unknown to the patient. She never had been assaulted, nor had any such case impressed her. She never had had any disease or injury of the genital region or of the breasts, nor to her knowledge had any of her relatives or friends had any such trouble. The same applied to operations in these spheres. She did not remember reading anything along these lines, nor had any one told her disconcerting tales of the nuptial bed. But

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when finally I asked her if she had ever seen pictures that impressed her, she quickly recalled that when about 11 or 12 years old she had seen in a foreign gallery paintings depicting the breasts of women being slashed, the nipples seized with red hot pincers and the like. These had made a deep impression on her. And at just that time her mother had talked to her and her sister about the horrible consequences of masturbation; had commanded and besought them never under any circumstances to so much as touch the genitalia. Here lay the foundation of the hidden fear and repugnance; a fear which had existed for years, of which she was quite unconscious, but which still was effectively potent. Treatment was brief because recovery was rapid.

Of this type are many cases of agoraphobia or claustrophobia. The patient either does not know he has a fear or he knows only that he has an ill defined apprehension. What he complains of is simply that in certain places or kinds of places he is exceedingly nervous and has certain alarming sensations and physical signs. The principal ones are tremor (or a feeling of internal tremor), palpitation, precordial oppression, epigastric distress, great weakness, cold or hot perspiration, flushing of the face, "rush of blood to the head," and a feeling of dizziness and dyspnea (sometimes with tachypnea). Frequently great emphasis is laid on gastric or abdominal symptoms, and not infrequently the patient complains of uncertainty on the feet or staggering, which does not exist. Oftentimes such a patient when asked what he is afraid of promptly responds that he is not afraid of anything. Indeed, to deny fear seems to be almost as instinctive as that emotion itself. He may deny it with indignation, asserting that he is not afraid but that he is uncomfortable and sick. A couple of cases will illustrate.

A woman, aged about 60, complained of nervousness, palpitation of a peculiar dizziness or lightness in the head. She felt so uncertain of herself that she could not cross a large room unless some member of the family took her hand. Her most intimate friend lived just across the street, but she could not cross to see her. She had valiantly tried. She would hurry down her own front steps and start with a

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rush for the other side of the street, only to stop at the curb as at a stone wall, held by some force which she did not understand. She could walk along the curb to the corner, but the moment she tried to cross, she had a peculiar sensation in the head, precordial distress, became weak and trembling, and simply could not put her feet further. The thing was a mystery to her, but it did not take very long to ascertain that an attack of dizziness some weeks before, the sudden death of a friend from heart disease, and the cerebral apoplexy of another had combined to fill her with a fear that she might suddenly drop dead. The fear once definitely exposed, treatment was simple and quickly successful. Perhaps it is worth while to note the procedure. She was taken to a hospital, saw no visitors, first took short walks with a nurse beside her, and then with the nurse 20 or 30 feet ahead. Soon the nurse walked a short distance behind her, then on the opposite side of the street. Following this the nurse would stand on a corner while the patient walked to the next corner and back. When she could do this with complete ease and comfort, the nurse waited while the patient walked around the block. The rest was easy.

An active and successful business man was unable to cross the open space or common between Michigan Avenue and the station where he daily took train to go home. He always waited on the curb until other people were crossing. When he tried to walk the 150 yards or so alone, palpitation, precordial oppression, a queer feeling in the head, and great weakness stopped him. He knew that he was physically sound, and recognized his disability as irrational; he said he felt as if some one ought to give him "a good swift kick." But he had not definitely recognized his trouble as a fear that he would drop unconscious while crossing the open space, with no one near to help.

A few years ago I was asked to examine a man, aged about 40, with "paroxysmal tachycardia." I rather demurred, saying that heart cases were out of my line. But I did examine him and could find nothing abnormal. Nor did any amount of exercise through which I could put the patient cause tachycardia. Finally close questioning brought the statement that he could walk any distance at any gait without palpitation provided his wife were along, but that a leisurely stroll by himself would be sure to bring it on. At once I sent him out to try it, and he came back with a pulse of 140. Then for the first time I got at his fear which was the cause of the tachycardia.

Occasionally the physical manifestations are pronounced, although the patient knows perfectly well that a groundless fear is the cause. A good example is the following:

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A woman, aged 42, well educated and unusually intelligent, had had since the age of 6 an overpowering fear of thunder storms. At that age she had suffered "a stroke of lightning." She was running home for shelter from the storm when just as she opened the door a bolt of lightning struck very near and she fell through the doorway onto the floor. Whether or not she received any electricity she could not tell me, but is said to have been temporarily paralyzed. In any event she received a great shock, possibly of fright alone. The timidity engendered by this occurrence was fostered by the parents, who allowed the child to take refuge in the cellar or in a dark closet whenever a storm approached. This astraphobia increased with the years. After a time a thunder storm caused not only a feeling of apprehension, oppression, tremor and palpitation, but also nausea and later violent vomiting and retching. In the meantime the patient had developed grave organic heart disease. When she came to me the situation was serious, inasmuch as the strain of each attack of vomiting and retching was more than the heart could compensate. Here, then, was a case in which the physical effects of a phobia were actually dangerous to life, although the patient quite well understood the nature of her trouble. I may add that in this case the nervous symptoms, at least the nausea and vomiting, were entirely controlled by means of hypnotism.

As has already been intimated, properly to elucidate a case of phobia, manifest or latent, generally one must go back to the very first appearance of the symptoms and investigate the circumstances surrounding it. In most instances the mechanism is quite simple. The patient has had some distressing or peculiar sensation or some disability from adequate physical cause. This sensation he at once or (more often) subsequently correlates with some serious malady or calamity which in some way has been brought to his attention. It goes without saying that the patient has as prerequisite a hypersensitiveness or impressionability. Consequently, often a very small trauma suffices. From my cases I note as the initial disturbance: ptomain poisoning, tobacco poisoning (excessive smoking), dizziness or syncope from overheated and overcrowded rooms, alcohol poisoning, the weakness or trembling from typhoid or other acute illness or following confinement or an operation, heat stroke, discomfort from excessive eating, rheumatism, aural ver-

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tigo (quite a number), tinnitus, syphilis, nocturnal emissions, the shock and hemorrhage of initial coitus, cerebral thrombosis, migraine, the distress of hyperacidity or indigestion, uremic convulsions, night numbness or night palsy, and predormal shocks.

Some phobias are very like the antipathies harbored by many people (for instance, for cats), so well depicted by Weir Mitchell and others. Such phobias may be hard to account for, as are the antipathies, but some are relatively simple. The following case is unique in my experience:

A woman, aged 27, could not bear to look at the sun, moon or stars, and was most uncomfortable whenever she thought of them or heard them mentioned. She never went out of the house at night unless compelled to do so, and would not even look out of the window. On a bright day she preferred to stay in with the shades drawn. She had become so worried and nervous that life was a burden to her and she a burden to her friends. Whisky gave her some relief, and at times she indulged to excess. As may be supposed, she had always been sensitive and timid about many things. In addition, she had been a spoiled daughter until she married an indulgent husband. On this basis her trouble developed as follows: At the age of 10 she had been in a tornado which she thinks did not frighten her excessively but left her afraid of storms. A year later her mother in great agitation awakened her and told her to dress quickly as another tornado was coming. Apparently these events left her very timid as to Nature and afraid of the dark. At 22 she married and went to live on a ranch, the nearest neighbor being 15 miles away. Here she was lonesome and rather unhappy, evidently oppressed by the isolation, the vast stretches of lonely range and her own impotence. A year after marriage a man came along with a newspaper telling that Halley's comet was about to appear and detailing all the horrible catastrophes said to accompany its coming. This recital had the immediate effect of giving her a nervous chill. She went to her room and took a big dose of whisky, but could eat no supper and could not sleep that night. This fear of celestial calamity rapidly developed into fear of all heavenly bodies; a fear which would seem to belong to the middle ages, but which did incapacitate a rational human being of this twentieth century.

One particular fear I wish to mention, as it is not uncommon, but frequently is unrecognized, principally because of the reticence of the patients. So far

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as I know it has not been named, but might be called hierophobia. I refer to a fear afflicting clergymen in their public functions. Oftenest I have seen it in Catholic priests whose duties about the altar are more complicated and solemn than those of a protestant preacher in the pulpit. Another reason, I believe, for its greater frequency in the Catholic clergy is that they officiate fasting, and the primary attack has been induced by the faintness of a man working on an empty stomach and who perhaps has slept poorly, has been up most of the night, or has been smoking excessively. In nature it is just like other phobias. For some casual physical reason the patient experiences a faintness or dizziness, or a tremor or palpitation, or something which suggests to him that he might lose control of himself and do something unseemly or that he might lose consciousness or have a stroke. One patient noticed that in administering the sacrament his hands trembled. This begot two thoughts: first, that his parishioners might think he had been drinking (he was quite rubicund but very temperate); second, that he might blunder and spill the host or the sacramental wine. This patient, like most of the others, complained not of his fear but of general nervousness, tremor, poor sleep, head pressure, etc. Methodical and patient investigation was necessary to extract the kernel of his trouble and show it to him.

To consider the treatment of these cases forms no part of my present purpose, but I will allow myself two remarks. First: The remedies most frequently prescribed for these disorders are absolutely futile except for possible suggestive value. How can "tonics" or "sedatives," change of climate, a vacation, tacking up a floating kidney, lifting up a prolapsed uterus or rest in bed and massage eradicate fear? Would a winter in Florida, a trip around the world, an operation for hemorrhoids, or strychnin pills make a sinner less afraid of eternal punishment? Second: The way to remove fear is to show the patient what it

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is and then teach him to demonstrate to himself that it is groundless. In short, educate him out of it as he has been inducted into it. If a child is afraid of a dark room we do not give him soothing syrup and tell him there is no room and no darkness. Nor do we suddenly throw him into the place of dread. We explain the darkness and the entire absence of anything to be feared. Perhaps we light the room and then make it dark again. Then, when we are sure he is ready for it, we take him kindly but firmly by the hand and lead him into the dark room, or perhaps only to the door or part way in. Finally, he goes in alone, and when he has fully demonstrated to himself that there is nothing there to be feared, of course he is not afraid and he is cured. We are only children of a larger growth. And not so very much larger either.

25 East Washington Street.

ABSTRACT OF DISCUSSION

DR. FRANK PARSONS NORBURY, Springfield, Ill.: I am a bit disappointed in Dr. Patrick's paper in that he did not go into the psychology of fear more extensively. I always look on this subject as one belonging to psychobiology, involving questions of survival, self-preservation, primary emotions and instincts. If we look at it from this viewpoint it does not matter what is the fear, but the fact that that fear exists. And when you analyze fear on this basis, even without the details, and consider the essential, instinctive, inborn condition that is present, I think we will then understand the whole fear habit of man. Of course, we must ascertain what is the fear. The question of survival is always paramount and the individual is put on the defensive by the emotion of fear. True, that is according to what fear stimulus may exist. We should consider that fear is not limited to any one condition, but that it is a tendency, like all physical traits, involving all of us alike. Just as hunger is not limited to any particular desire for food of a certain quality, but the question is that we desire food, and this involves all centers and all cells. And so it is in fear. When we have fear the reaction is fear alone and involves the stability of all cells. It is therefore a question of biology and in consideration of the subject fear we should start from that viewpoint.

Dr. Patrick apologized when he mentioned the freudian doctrine. To me this is an essential question. In my judgment it is more than Freud's emphasis on the sexual instinct.

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We should call it the freudian wish, in which is involved the essential determining factor of life itself, at least in life preservation. The question is one requiring full elaboration of all the psychobiologic phenomena. And for that reason, when we take into consideration the fact that we are dealing with primary emotions and instincts, with the laws of survival, we have a rational basis for procedure. There may be an association test that will strike real fear, the elements that are concerned in the foundation of the complex, because fear is the root of many of our intellectual strivings. It is the strife which brings about adjustment and when you know that, you are readjusting and reeducating the personality. The essayist speaks of educating the child to go into the dark. If you get down to the primary analysis of why the child is afraid of the dark and show that it is an element of survival and of self-preservation, and then treat the question from the standpoint of what are the primary instincts, you will by this means get a better answer. You have to make an explanation. The child is seeking an explanation; he is looking for an answer to his instinctive reaction to his fear, just as you or I would in any other reaction. This is normal; it is a stage in development and an onward movement in individual development of personality. The fact of the policeman being not afraid to meet the criminal alone only has value as an evidence that he has adjusted himself to such fears. This is the biologic consideration of the question. The other reaction to fear is only of importance because of the fact that he has a fear.

Dr. Harold N. Moyer, Chicago: Dr. Patrick said that it was a work of supererogation in this organization to separate impulse and fear and explain the distinction. I do not believe that is true. He tells us that the freudian doctrine is not essentially sexual, but here we are confusing the two things again, fear and impulse. And that is the point. We are afraid of the freudian doctrine, we fear that it is complicated by a sexual impulse. We do not want to be charged with the latter, hence we are afraid of the former. That is the doctrine. I suppose that within the month hundreds of people are sent to institutions for the insane with the statement that they are not only insane, but that they are suicidal. What is the basis of that statement made in their committal papers? Why, they have confessed to the doctor or to the examining magistrate or to somebody that they were afraid they would kill themselves. But they never intended to kill themselves—they were *afraid* they would. It was simply a morbid fear, they were not suicidal at all. A man quietly puts away his razor for fear he will kill somebody, hence in the papers regarding commitment he is put down as homicidal because he is *afraid,* he says. That is the common misconception—the relation of fear and impulse.

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DR. ROBERT MCGREGOR, Saginaw, Mich.: The emotion of fear must always naturally arise from an instinctive basis. Its reaction is as forceful and uncontrollable as a reflex. I believe that fear of a vague and unreal kind plays a potent part in many of the manifestations of hysteria. For example, I knew of the case of a strong, husky farmer who stiffened out in a tonic spasm while the dentist was applying the rubber dam. Two days later he returned, reinforced by a couple of his neighbors, when he went through the ordeal without a hitch. The remedy in the young subject is disciplinary and educational, along the lines of rendering more stable the emotional equilibrium.

DR. WALTER B. CANNON, Boston: The emphasis which Dr. Norbury has placed on the biological aspects of this discussion is well deserved because of the fundamental character of these emotions and the instinctive impulses that attend them. Dr. Patrick is, I believe, doing an important service in calling attention to the significance of these fundamental experiences which human beings share in common with the lower animals. During the past four or five years we have been studying the effects of fear and of aggressive feeling in lower animals, and the results obtained have rendered the suggestions which Dr. Patrick made today particularly interesting to me. He pointed out the very close association between courage with readiness for aggressive action, and a fear which likewise came on with great intensity. If you study the reactions that occur in lower animals either from fear or from intense aggressive feeling, you will find that they are essentially the same. All the changes noted—increased heart beat, inhibition of the movements of the stomach and intestines, liberation of sugar from the liver, increased blood pressure—occur either in fear or in rage, so that basally the biologic changes are alike. And there is good reason why they should be alike, because in either case, whether there is fear present because of danger against which the organism has to defend itself, or whether there is aggressive feeling which will result in attack, the organism has to engage in a struggle, a struggle which may mean life or death to it and consequently will call for the mobilization of all its forces. So there is abundant reason why, under the circumstances referred to, these changes, all of which are favorable to the mobilization of the abilities and activities of the organism, should then occur as reasonably in fear as in anger or rage. James years ago pointed out the very close relation between fear and anger. He remarked that we both fear and wish to kill anything that may kill us. And it seems to me that if we regard these emotions and the instincts that are associated with them *biologically*, and apply the biologic concepts to the peculiar instances and circumstances that may arise in particular cases, we may often get an illuminating insight into their actual operations.

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DR. TOM A. WILLIAMS, Washington, D. C.: There is great confusion in this whole question. Of course, fear is a biologic reaction; of course, there is a physical mechanism; of course, we cannot arrest it by attacking the physical mechanism. Let us analyze the matter. There is first a reaction, what we call the manifestations of fear, physical and psychologic. The cause of that in all cases from the lowest biologic organism up to the human being is a sentiment, a feeling, an apprehension of danger. The cause of that is a concept always. Because of that concept there is a percept of the situation. It is because of what we fear that makes us fear. Then it is only by reason of the removal of the primitive active factor of the fear that we can remove the fear. We cannot remove the fear by dealing with the ultimate mechanism. Therefore, the question is a psychologic one, and we must find the active percept which in each particular case produces fear. But the same situation does not always produce fear in every individual. Why? Because in each there is a different set of conceptual processes causing fear, and it is those which we have to attack by exploratory analysis. The only exploratory analysis is not freudian analysis. People were cured of these troubles long before that doctrine was known.

Dr. Cannon has shown how it is the concept which determines fear in the lower animals. All of us who have taken the opportunity to examine committal cases have found this to be true, and the mechanism by which the deviation from normal reaction occurs we can call what we like. I call it suggestion, which simply means a false appreciation of the situation, a lack of placing the situation in its true relationship to life as a whole and to the individual himself. It is true that certain individuals are more apt to deviate in that way. We know that certain individuals are nervous, easily intimidated. One reason is the physiologic tendency toward the fear reaction, and the other is the environment. And it is this that we can, perhaps, influence. We can get at the way in which this individual reacts by going back to the childhood influences and environment, and readjust the mental attitude toward life, by this means suppressing at the inception the reactions which would otherwise eventuate in fear. Many cases of that kind have been published to show that these matters can be readjusted quite independently of the methods of the freudians. It is only because the patient cannot explain all the things that create the fear. It is not that he does not know that it is fear. He does not call it fear; he does not recognize the similarity of the reaction to his fear, but when placed face to face with the psychologic features of his own case he recognizes them as not derived fundamentally from fear. Hence even in Dr. Patrick's cases I believe there is no really unconscious

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mechanism, in which respect I think we can part company entirely with the freudians.

Dr. Walter B. Swift, Boston: I have done work along this line in connection with stuttering. In cases of stuttering we have found that a great many fears precede the utterance. Those fears are manifested through the utterance where stuttering is present, and many times follow afterward. The treatment that has been applied very successfully to these cases is what is called the supplying of a visual image. By this I mean that during the stammering process the visual image, that is normally present in talking, is absent. Therefore, by training these stutterers to visualize automatically during talking, their fear vanishes and their stuttering improves. I think that these fears in a general sense often come from false instructions and from interpretations of older people who have drilled them into us in our early days. Therefore it would seem that the general method of eliminating fear in these cases should be to supply automatic visual images. Cure results.

Dr. C. B. Burr, Flint, Mich.: It is true that those patients fear the unfamiliar, as Dr. Patrick has said, but they also fear the familiar, as witness syphiliphobia. A distinct delusional state occasionally is determined by harking back to a genuine or supposed infection.

Dr. Foster Kennedy, New York: I want to point out something which in the practice of psychanalysis comes under one's observation in the day's work: That the whole theory of symbolism as enunciated by Freud, whether or not it be true, in the practice of the disciples of Freud, is used as mechanically as would be a set of chemical analytic tables. The details of Freud's symbolistic theories are as ex cathedra as are the enunciations of Mary Baker Eddy, and are accepted equally implicitly by the followers of the two systems of thought. In practice this has led not to the careful detailed personalized examination of the individual under consideration, but to a request for symptoms and their automatic pigeon-holing on a basis of preconceived data of purely pontifical character.

Dr. Francis Rhodes Fry, St. Louis: There is no place for the freudian intrusion in any serious sense in the practice of medicine and psychiatry. It has been my observation that a phobia is often sustained by some point of irritation or distress in the patient's periphery, so to speak. In that class of cases, particularly, it does not do to remind the patient too soon that his trouble is psychic.

In a recent paper on the anxiety neuroses, the peripheral as distinct from the psychic side of these cases was my chief theme. Very often the fear patient has an anxiety based on a peripheral irritation constantly suggesting to his mind that something is wrong somewhere in his system. It is well

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to work seriously to correct possibilities of this kind. In other words, to begin something that looks like a campaign in the patient's interests. I have seen many patients affronted and driven away by the first interview with a practitioner, who has simply said, "Nothing the matter." Of course, such practitioners are generally not good enough psychiatrists to grasp a situation of this kind.

Dr. Charles R. Ball, St. Paul: I think we can say of the freudian doctrine that it has some elements of truth, but because of these few elements of truth it is not necessary for us to be compelled to swallow the whole thing. I believe absolutely in Dr. Patrick's and Dr. Williams' exposition of this subject: That correction must be along the lines of readjustment of the patient's ideas. With these patients we should do as in other affairs of life, "Show them." And still that is not all, because there is a biologic factor. It does not appear so plainly in the nervous cases as in the mental. I remember the case of a man with manic depressive insanity and with a fear of suicide. When I saw him his tongue was furred, he was losing in weight, there was lack of appetite, disturbed sleep, and he went on getting worse until one day while sitting in his office he did attempt suicide. He made a vicious slash at his throat, cutting himself from ear to ear, and slashed the arteries of his wrists, but fortunately did not kill himself. From that time on his fears left him, his tongue immediately cleared up, his sleep came back, and mentally he was himself again. This case illustrates the biologic side—that there is something which brings about those conditions in addition to ideas. I can recall a number of cases of this character where some great shock or perhaps some other fear has immediately relieved a manic depressive condition and suicidal tendency.

Dr. Ulysses Grant Darling, Chicago: It appears to me that in the elimination of fear, whether it be in animal or in human being, the greatest point in the cure is to give the individual the experience of the feeling of safety. It is not always necessary to be so very scientific or to follow after any particular hypothesis in the analysis of the individual. It also appears to me that the doctor, whether from a scientific standpoint or from an artistic standpoint, who is equal to the occasion at the moment of interview of discovering a method of giving his patient this experience, will have gratifying results. In the analysis of any morbid fear it must be an analysis that reaches that particular fear, and then we must be capable of giving the treatment which, after a clear-cut diagnosis, will have a particular bearing on the case in hand.

Dr. David S. Booth, St. Louis: I am unable to reconcile the view of the essayist on the treatment of fear with a fear which I will have to personally acknowledge in order to

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explain it. I have a fear of snakes and lizards, which I know is unnatural, although I realize that there is no reasonable basis for this fear. When a boy I had a classmate who carried them in his pockets and played with them and I know from knowledge of natural history that there are very few dangerous snakes. Even the imitation wooden ones make me "creep," and I also had that kind of "snaky feeling" when I used the ordinary earthworm used in fishing. Nothing of the kind has ever attempted to harm me, I have never been frightened with those things in any particular manner only in running onto them as boys naturally will in play. But I will admit that I can hardly look at a snake or lizard until after the initial feeling of fear has passed off, when I am able to kill it. I would like to have some freudian explain why I cannot get over this feeling.

Dr. Hugh T. Patrick, Chicago: Dr. Aikin has handed me a written suggestion, as follows: "Please emphasize the value of personality and our complete analysis of each patient as an individual and not a machine, as one in 10,000 made in the same mold." I cannot make it any more emphatic than that. To Dr. Norbury, I can only say that because I omitted to discuss the psychology and the biology of fear, I do not wish to be understood as not being entirely in accord with him and Dr. Cannon. In my paper I speak of fear as one of the fundamental things in us, and I illustrate the fact that we are controlled by our primitive emotions and appetites; not by reason, not by our intellectual processes at all. In a way I have said that there is some physical cause to start this thing. Of course, that might have been a transient physical something or it may have been a continuous physical something, and if there is a continuous physical something which the patient fails to interpret properly, naturally it perpetuates the fear. And if we do not find out his error and correct it we have not done our duty. Then there is the next step, which Dr. Darling also alluded to, and which I should like to put perhaps very crudely in this way. We can scarcely be said to treat a psychoneurosis; we are working with the individual who has it. And I believe that in some instances the object should be—I say this with great deliberation—the object should be, not to give the patient the true explanation, which is to be preferred and in the majority of cases is the better procedure, but to give him an explanation which satisfies him; which appeals to his particular mentality, to his particular viewpoint, which harmonizes with his past, his education, his environment. And I thoroughly believe that some of the successes of the freudians have been attained not because they have found the truth, but because they have done something which begot confidence, which presented to the patient what for him was an adequate explanation. If

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we can make the patient *sure* that we understand, and if we can make him believe that he understands, it does not really make any very great difference whether we have reached the bottom of the psychology of fear in that individual or in the race—we have accomplished our object with that individual patient.

One symptom spoken of was the vague indeterminate sensation of fear. What I have tried to make clear is that most of these fears are not vague and indeterminate at all; they are perfectly definite, individualized fears, each one of which we can get hold of and bring to the surface so that we and the patient himself can recognize it.

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SYPHILITIC PSYCHOSES ASSOCIATED WITH MANIC DEPRESSIVE SYMPTOMS AND COURSE

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The routine application of the Wassermann test and the study of the cerebrospinal fluid have shown that, apart from diseases of well marked syphilitic pathology, there are cases which clinically course in ways not usual for the syphilitic types of psychoses, although showing serologically the pathologic changes of that disease.

A considerable number of cases may show positive Wassermann reaction in the blood and no pathologic condition of the cerebrospinal fluid. The majority of these are instances of more or less marked types of non-syphilitic mental disorders, which show an incidental constitutional syphilis. A smaller number are forms of syphilitic psychoses of a type other than general paralysis.

There are other cases which show syphilitic changes in the blood and cerebrospinal fluid similar to those found in general paralysis, but which in their clinical symptoms and course seem to stand apart from this disease and present interesting points for differential diagnosis. Such are the cases which course clinically with the symptomatology and cyclothymic variations of manic depressive insanity. The occurrence of this type of case has occasionally been commented on in the treatises on general paralysis, in which they have usually been regarded as combinations of general paralysis and manic depressive insanity.

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I have been able to collect a number of cases of this type from the records of the State Psychopathic Hospital at Ann Arbor, and for the others I am indebted to the Pontiac State Hospital.

The following brief abstracts are illustrative of this type of case:

CASE 1.—*N. B. H., woman, aged 39. Since 22 short attacks of depression with norbal intervals. At 35 severe excitement of the manic type, with extreme irritability continuing without interruption for four years; very slow intellectual deterioration; few neurologic disturbances. Strong Wassermann reaction in serum and spinal fluid. Pleocytosis.*

Little is known of the patient's history before her marriage at the age of 22. Her father is said to have been demented at the age of 65.

Three months after her marriage, she became despondent, accused her husband of not caring for her, and attempted suicide. After a month she recovered completely. Since then she has been commonly mildly depressed before her menstrual periods, and has at these times talked of suicide. She never became pregnant.

In 1910 her husband contracted syphilis, but she herself never had any external evidences of the disease. In April, 1912, she became more irritable toward her husband. She accused him of misconduct and spoke of attempts he had made to poison her.

In May, 1912, she was admitted to the State Psychopathic Hospital. She was then much excited. She talked loudly, and her remarks concerned the attitude which various persons had taken against her. Many of her ideas were of sexual significance. "She had been drugged and thus brought under the influence of others." She comprehended questions, but her attention was not held to the question, and she rambled off into a flight of ideas, made up largely of her accusations, and suspicions, and expansive references to herself.

Her conduct was extremely boisterous and aggressive against whatever was done for her. In her actions there was often an element of playfulness. Her stream of spontaneous thought showed numerous influences of incidents in her environment and of sound associations. At times she decorated herself fantastically. There were several episodes in which auditory hallucinations occurred. Her conduct became violent in the extreme, and she had no regard for personal decency.

Her excitement made a systematic physical and neurologic examination impossible. The pupils were unequal and irregular in outline. The light reaction was present. There was a slight tremor of the tongue. The knee jerks were active and equal.

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This excitement continued until the spring of 1913, when she gradually became quieter. A more complete examination at this time showed unequal pupils and lessened reaction to direct light. The tongue deviated to the left. The knee jerks were exaggerated.

During the summer her conduct was such as to allow her to be about the ward, and she did some work. Often there was an element of playfulness in her actions. Delusional ideas were often expressed.

The cerebrospinal fluid in September, 1913, showed 77 cells per cubic millimeter. The albumin was about normal. The Wassermann test was three plus. The blood serum at this time was four plus.

Since then, for the past two years the patient's condition has been one of extreme excitement. There has always been a marked irritability which easily leads to assaults and destructiveness. She has no sense of decency, and in her personal habits is filthy in the extreme. She talks much in a very boisterous manner, and her thought is distractible and influenced by sound stimuli, and is that encountered in severe degrees of manic excitement. At times she will control herself and converse in an orderly manner until irritated by some remark or incident, when it will lose its coherency. Many of her actions show a purposeful character and are directed by impulses toward mischievous and malicious behavior. Her thought, as a whole, is less coherent than formerly, but her perceptions and comprehension are quick and clear.

Her memory for events from day to day and for recall of the past is good, and aside from the degraded character of her conduct, deterioration has not advanced much during the last four years.

There is no speech defect, and the knee jerks are active.

CASE 2.—*A. S., woman, aged 31. Indefinite attacks of "nervousness" until 31, when she became depressed for two months, then passed into a state of typical manic excitement. Few neurologic symptoms. Wassermann reaction strongly positive in blood and fluid. No pleocytosis.*

As far as known, the family history is free from nervous or mental diseases.

Meager information regarding her personal history states that at 17 she had typhoid fever lasting two months. She had always been subject to "nervous spells," and has suffered much from neuralgic pains all over her body. She was married at 19 and has had five children, one dying of diphtheria.

For years she has pains and excessive flowing at her menstrual periods. One month before her admission, an operation was done on the uterus to benefit the excessive flowing. She was convalescing well, when one of her children fell down stairs, and she became much upset. Her mood became continually depressed, and she worried much over her finan-

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cial affairs. She attempted suicide, and two weeks after the beginning of this depression, she was committed to the Pontiac State Hospital, in October, 1915. The examination showed that she was under weight, and there were a few râles heard in the right axilla. Heart and urine were not abnormal.

Neurologically, she told of having had neuralgic pains for a few years, which were worse at night, and attacks of dizziness. The pupils were regular, but the direct light reaction was absent. The knee jerks were much increased. At times during the examination she became agitated and there was some trembling. Her speech was without defect in articulation. The handwriting was done slowly and was rather irregular and tremulous.

The cerebrospinal fluid showed 5 cells per cubic millimeter. There were increased albumin and globulin. The Wassermann reaction was four plus, and Lange's test gave a curve of 1 2 3 3 2 1 1 0 0. The blood serum was four plus by the Wassermann test.

Mentally, her attitude was one of mild depression. Her movements were slow and deliberate, and her speech was low and slow. At times she became mildly restless and apprehensive. Comprehension was clear and orientation correct. At times it was an effort to hold her attention to the question, and she readily became confused. This was quite marked in her attempts to do simple calculations. The memory was fair for recent and remote events.

Her mood was mildly sad. There was a subjective feeling of illness and that her nerves had got beyond her control. Her thought content was chiefly related to her interest in her health, and there were occasional somatic complaints, such as "a feeling of pressure" which she referred to her epigastric region, and this suggested to her that probably she had a growth around her heart. There was some self-criticism, as "I wish I could go back and do what was right by them." Her conduct was at all times correct. She slept badly, and at nights her restlessness was more marked.

She improved rapidly, and about a month after her admission she was cheerful and active, working interestedly, and could not understand why she had held the sad ideas that had been present during her early stay in the hospital. She was taken home and remained away for about six weeks, when she again became depressed and was inclined toward suicide, and was brought back to the hospital, Jan. 18, 1916. She was then about as she was during the first weeks of her former admission; mildly sad, with somatic complaints, and worried unduly over her affairs. She improved rapidly, and a month later was again taken home. At first she did well, and then became suspicious of her husband's conduct. She sought the aid of the police to protect her. At first mildly restless, she later became destructive to things about the home, and, May 11, 1916, she was again returned to the hos-

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pital. She was then overactive, laughing loudly, whistling, and boisterous in her conduct. Her mood was elated, and there were ideas against her husband. Since then her attitude has continued typically manic in its character. She is overactive, turbulent and aggressive. Her mood is elated and she shows a very marked flight of ideas, with superficial associations. At all times she is quick and clear in her comprehension and orientation.

CASE 3.—*J. C. B., man, aged 64. Syphilis at 19. Periodic attacks of elation and depression of short duration, with normal intervals throughout adult life. At 64 depressed. Neurologic disturbances. Depression passing off, leaving a very mild deterioration. Wassermann reaction strongly positive on blood and spinal fluid. Pleocytosis. Increased albumin.*

In the patient's family, a brother and a sister had been insane. At the age of 15 he is said to have had an attack of cerebrospinal meningitis, which destroyed the vision of his right eye. At the age of 19 he contracted syphilis.

His past history shows the occurrence of periodic attacks of elation and depression at various times during his life. One attack occurred at the age of 48 and lasted about six weeks. One at 49 lasted two months, and one at 50 lasted five months. Other attacks occurred at intervals until his admission to the Pontiac State Hospital at the age of 64.

The important neurologic findings at that time were: blindness of the right eye: Argyll Robertson pupil in the left eye; all of the tendon reflexes slightly exaggerated. There were no tremors or incoordination. The hearing of the right ear was diminished.

Mentally he was clear in his comprehension and orientation. The memory was impaired for both retention and reproduction. His mood was one of a slight depression with mild apprehensiveness concerning his physical condition. Aside from a moderate slowness, there were no thought disturbances.

The cerebrospinal fluid showed 25 cells per cubic millimeter. There were increased albumin and globulin. The Wassermann reaction was three plus on the fluid and four plus on the blood.

His depression continued extremely mild. He showed interest in work, but there was always present a certain lack of spontaneity, and the memory defect was pronounced.

Two years later the depression lifted, but on account of his mild deterioration he remained in the hospital and finally died from a railway accident.

CASE 4.—*H. E. B., man, aged 33. Syphilis at 19. At 23 short attack of manic excitement. At 25 manic attack, followed by depression and recovery. Slight neurologic disturbances. At 28 manic excitement followed by depression and recovery. Wassermann reaction strongly positive on blood and spinal fluid. Pleocytosis.*

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The family history so far as known was negative. In his youth the patient was regarded as irritable and difficult to control, and in his school work he was backward.

At the age of 19 he contracted syphilis. At about 20 he was noted as being full of schemes, visionary, and unstable in his conduct. At 23 this had increased, and he became so excited as to be committed to the Pontiac State Hospital. There, he was overactive, distractible in his attention and thought, and exhilarated in his mood. After about three weeks he had become quiet enough to be discharged.

In August, 1908, he became overactive and exhilarated in his mood and was again sent to the hospital. At that time he showed slight unsteadiness in Romberg's position, and there were fine tremors of the face and hands. The pupils and knee jerks were within normal limits.

Mentally, he was clear in his comprehension. His memory was not impaired except when influenced by his distractible thought. He had a typically manic flight of ideas, and was overactive and impulsive in his conduct. After three months, the excitement subsided and he passed into a state of mild depression, with retardation of thought and movements. This passed away, and six months after his admission he returned home.

In 1914 he was again admitted to the hospital in a severe degree of manic excitement. The pupils were not abnormal. The knee jerks were exaggerated. There was no speech defect. Three examinations of the cerebrospinal fluid showed cell counts varying between 24 and 35 per cubic millimeter. The albumin and globulin were not increased. The Wassermann reaction was in each examination three plus, and always four plus on the blood serum.

Mentally, he was clear in his comprehension, but overactive and exhilarated, and showed a marked flight of ideas.

Under intraspinal treatment the number of cells of the cerebrospinal fluid fell to 2, and the strength of the Wassermann reaction was lessened. His excitement continued for two months, a depression then occurred which listed six months, when he again had improved sufficiently to be allowed to return home.

CASE 5.—*A. G. L., man, aged 40. At 24 manic excitement, brief duration, recovery. At 36 manic excitement, brief duration, recovery. Similar attacks at 38 and 40. Neurologic disturbances. Wassermann reaction strongly positive on blood and spinal fluid. Pleocytosis.*

The family history stated that both his father and mother were insane. There was little information as to the patient's personal history until after his marriage at the age of 24. At about this time he had an attack of excitement. For some time previous he had been a little more restless than usual and had drunk heavily. He was sent to the Pontiac State Hospital, where he showed mild restlessness and pressure

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of thought. He rapidly improved and was discharged recovered four months after the beginning of the attack.

He was well until the age of 36, when he became irritable, getting into difficulties with his family and the neighbors. He was again admitted to Pontiac. There he was restless. His mood was euphoric and his ideas were expansive. He was clear in his comprehension and orientation. The pupils were normal in outline and reactions. The knee jerks were decreased. There were no disturbances of handwriting. After three weeks he had gained control of himself and left the hospital.

He remained well until he was 38, when he again became unstable and overactive, and was brought into the hospital. After a few weeks he was again well.

At the age of 40, he had another attack of excitement and was again in Pontiac. His excitement was somewhat greater than on previous occasions. He was clear in his grasp and orientation. His attention was distractible. He was noisy, singing and talking, with a marked flight of ideas. The pupils were not abnormal, but both knee jerks were absent. There was a slight Romberg. The blood serum gave a four plus Wassermann reaction, and the cerebrospinal fluid showed normal albumin reaction, 25 cells per cubic millimeter, and a four plus Wassermann reaction.

The blood serum of his wife, and son aged 9, both gave a four plus Wassermann reaction. His thought and conduct were typically those of manic excitement. After seven months he was again discharged recovered.

CASE 6.—*W. C., man, aged 49. Syphilis at 23. At 26 brief attack of depression. Since then annually brief attacks of depression. At 49 depressed. Neurologic disturbances. Wassermann reaction mildly positive on blood and spinal fluid. No pleocytosis. Recovery from mental symptoms. Improvement in serologic findings.*

The family history stated that his grandfather suffered from two attacks of depression. The father showed eccentricities of conduct.

As a youth he had a period in which he had fears and compulsive ideas. At the age of 23 he became infected with syphilis. At 26 while taking his final examinations in a dental college he had an attack of mental depression which lasted about a month. Since then, he has had at least once a year a period of depression lasting several weeks. These usually were associated with a phase of mild elation.

In March, 1914, at the age of 49, he became depressed over business affairs and developed ideas of self-accusation, and had suicidal inclinations, which brought about his commitment to the State Psychopathic Hospital at Ann Arbor.

There were then present irregular and unequal pupils, and dulled reaction to light stimuli. The knee jerks were unequal. The left Achilles reflex was only occasionally obtained.

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There was slight incoordination of movements, and unequal innervation of the muscles of the face. There was a slight articulatory speech defect. The cerebrospinal fluid in two examinations showed 3 and 4 cells, respectively, per cubic millimeter. The albumin was not increased, but the globulin was a little more than normal. The Wassermann reaction was present in a strength of two plus on two occasions. On the blood serum the reaction was at first one plus, and after treatment it became negative.

Mentally, the patient was inactive, although not slow in his movements or speech. He was always depressed, and his thoughts were sad and centered on his health. At times he expressed suicidal inclinations. There was marked impairment of his memory. He quickly forgot numbers and what he read. Intellectual work was difficult and often incorrect. A week after his admission the depression was less and he returned home.

A few weeks later he returned to the hospital as deeply depressed as at any previous time. He expressed many absurd somatic ideas. At times he was dull and confused. After three months, the depression became less. He was always clear in his comprehension, and his intellectual ability and memory improved. He was at this time discharged, and for eighteen months has been successfully conducting a large dental practice and is apparently free from all mental depression.

CASE 7.—*B. E. S., woman, aged 31. Syphilis before 26. At 30 attack of hypomania followed by depression; mild neurologic disturbances. Wasserman reaction strongly positive on blood and spinal fluid; pleocytosis; marked improvement, remaining nearly recovered for three months, then apoplectiform attacks; rapid deterioration; excitement of manic type, followed by depression, and death from exhaustion.*

The family history showed that the patient's mother was insane, the diagnosis being manic depressive insanity.

At the age of 26 she had paralysis of the right sixth nerve, with headaches and dizziness. These disappeared under treatment. At 30, following an ovariotomy, she became overactive and expansive in her ideas. This continued for a year, when she became depressed, cried much, and attempted suicide. After a short period of improvement, she passed over into a deep stupor, in which she neither spoke nor moved, and in this condition she entered the Pontiac State Hospital. The neurologic examination at that time showed paralysis of accommodation in the right eye. The knee jerks were exaggerated. There were tremors of the hands and tongue.

The spinal fluid showed normal albumin and slight presence of globulin. There were 65 cells per cubic millimeter. The Wassermann reaction was four plus on the fluid and on the blood serum. The patient was put on mercurial treat-

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ment, and six months after admission she was regarded as practically normal, and was discharged.

Three months later she became overactive and was returned to the hospital. She was then clear in her comprehension, and there was no memory defect demonstrable. She was restless and overactive, and her thought was flighty.

Ten months later she had a series of apoplectiform attacks and one convulsion. Mental deterioration progressively became more prominent, and there was a pronounced speech defect. This excitement continued for eleven months, when she became depressed and stuporous. Death occurred from exhaustion about two months after the beginning of the stupor.

The brain in this case showed an extreme thickening of the pia mater with a large number of infiltrating mononuclear cells. There were extensive infiltrations of the blood vessels throughout the cortex and pons. The nerve cells and neuroglia were less severely affected than the membranes and vessels, yet there were many evidences of changes seen in the usual process accompanying general paralysis. The aqueduct of Sylvius was much narrowed by a proliferation of the subependymal glia, and this brought about a marked dilation of the lateral ventricles.

In the foregoing histories there is a certain recurrence of attacks which emphasizes the resemblance to manic depressive insanity. There are other instances in which in a single attack the symptoms closely resemble those seen in that disease. These usually are psychoses in which mental depression with slowness of thought and delusional elaboration dominate the clinical picture.

CASE 8.—In the family of E. L., man, aged 44, there were several instances of death from apoplexy, but otherwise no important abnormalities. The patient had syphilis at 28. At 29 he had an attack of anxiety with mild depression lasting for two years. He had no more attacks until he was 44, when following a mild attack of syncope he became agitated and depressed, and entered the State Psychopathic Hospital at Ann Arbor.

The physical and neurologic examinations showed a lesion of the aortic opening; chronic nephritis; irregular and unequal pupils, and impaired reactions to light. There were no tremors or speech defect. The Wassermann reaction on the blood and on the spinal fluid was two plus. There were 18 cells per cubic millimeter, but no increase in albumin or globulin.

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Mentally, the patient was profoundly depressed. His thoughts were centered on his nervous condition. There were a few somatic delusions. The memory and intellectual capability were not impaired. There were marked insomnia. Under treatment, the serologic findings improved, but his mental condition has not changed, and now, three years after his admission, he is still much depressed and unable to apply himself to profitable work.

CASE 9.—The mother of O. S., man, aged 32, died of general paralysis, and a brother died in an attack of mental disorder. At 25 he contracted syphilis. At 30, following the death of his mother, he became anxious regarding his own health. Examinations showed a strongly positive Wassermann reaction on his blood. He was extremely apprehensive, and was continuously mildly depressed.

In 1913 he entered the State Psychopathic Hospital. At this time the knee and patellar reflexes were absent. The pupils were irregular but reacted to light. There was Romberg's symptom and slight ataxia and tremors. There was no speech defect. The Wassermann reaction was strongly positive on the blood and fluid. The cells have varied between 28 to 8 per cubic millimeter.

His depression has disappeared, and he is now working capably.

COMMENT

In all of these cases we have mental symptoms which differ much from the usual clinical picture of general paralysis, and seemingly are unrelated to the direct effects of a syphilitic process involving the brain structure.

Clinically the cases of the first group in their symptoms resembled common types of manic depressive insanity, in which there is no evidence of syphilis. In the majority of these, organic neurologic disturbances were not prominent. In only two cases was memory disturbance at all marked. In but one case, a fatal case, was there any general mental deterioration.

It seems justifiable to regard cases of this type as coincidental occurrences of manic depressive types of reaction and syphilitic diseases of the type of general paralysis.

The relation between the two disorders might be made clearer if one knew how far back in the course of the disease the serologic pathology became mani-

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fested. In only one case were there any prominent neurologic disturbances, such as attacks, speech defects, ataxia or reflex abnormalities noted during the early phases of the clinical course. In some of the cases there were signs of manic depressive tendencies before the infection with syphilis. In two cases the first attack of manic excitement occurred a few years after the infection. It thus seems impossible to establish the infection with syphilis as the direct cause of the mental symptoms.

The question of combined psychoses has always been interesting in psychiatric discussions. This interest has lessened as the psychiatric point of view has been turning away from nosologic distinctions, and toward regarding many of the functional psychoses as individual types of reaction to personal experiences. In this way the mental symptoms are only related to structural brain disturbance, so far as the latter introduces difficulties in nervous functioning and makes it possible for tendencies which have resulted from a pathologic nervous constitution, and the effects of personal experiences and mental habits, to assert themselves.

That in these cases there are possibilities for pathologic nervous constitution is shown in the frequency of hereditary instances of mental diseases in the families of these patients. These were present in seven of the nine cases we have been considering. In some of these families there were several members who had been insane, and in several instances the insanity present was of the manic depressive type.

Aside from the structural influence of a syphilitic process of the nervous system in producing disease, there is the possibility of a psychogenic mental disturbance which may develop in the individual who is confronted by the new and distressing problems which come to one who has acquired syphilis. It is not uncommon for functional mental disturbances to occur in the early stages of syphilis, the only explanation for

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which are the fears consequent upon infection. It is well known that manic depressive psychoses often have their beginning in a disturbance of the normal moods, brought about by a troublesome situation.

It is of interest to note that as far as the evidence of structural involvement of the central nervous system in this group by a syphilitic process is concerned, the progress of the process was slow and mild.

It would seem that some prognostic information might be drawn from the analysis of even these few cases. Of the patients, after the serologic examinations had shown the presence of a syphilitic process in the central nervous system, four made improvement in their mental symptoms to a degree that permitted them to return to their homes, and some to take up their business again in an efficient way.

One might conclude that cases of mental disease with the neurologic findings of general paralysis, when the mental symptoms are those of excitement or depression of the manic depressive type, in a considerable proportion tend to have very complete remissions, and their neurologic symptoms run a mild course.

ABSTRACT OF DISCUSSION

DR. FRANK PARSONS NORBURY, Springfield, Ill.: Some years ago, before modern methods of diagnosing syphilis were established, a patient came under my observation for treatment, having the clinical secondary manifestations of syphilis. He was admitted to the hospital as insane, manic—depressive form, and ran the usual course of manic symptoms and convalescence in that type of case, and returned to his home. The following year we ascertained some facts connected with the history. About two years preceding admittance to the hospital he had an attack not unlike that described as a somnambulistic state, he went to a hotel, in a distant city from his home, registered, saw his friends, and, after a time, came to himself in this hotel and returned home. He had three subsequent experiences of this kind, during which he went through this same reaction, going to the same hotel and then when coming to himself would return home. After passing through the manic-depressive period in the hospital, the following year he entered medical school where in his second or junior year while sitting

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on a bench one day in the clinic he had a convulsion and died. I have always been interested to know, especially in the particular case referred to, whether we were not then dealing with two essential conditions. One manic in which acute syphilis may be a factor; the other, some organic change independent of the syphilis. The syphilitic infection was subsequent to the first somnambulistic experience.

Dr. Hugh T. Patrick, Chicago: I would venture to expand Dr. Barrett's theme a trifle and apply it also to some of the neuroses, drawing the same moral: That if the patient presents a sufficiently typical clinical picture of a neurosis, and we get a positive Wassermann as evidence of syphilitic infection, we must not conclude that he has a syphilitic neurosis (if there be such a thing), or that the neurosis is caused by syphilis. It is the old clinical caution to consider all of the data and try to come to a reasonable conclusion. And I say this because I have had in a few cases an experience almost as uncomfortable to me as to the patient. For instance: A man with all the symptoms, or a sufficient number of symptoms, of ordinary psychasthenia, had a great fear of syphilis. I gave positive assurance that he did not have syphilis and that his symptoms could not possibly be due to syphilis and that there was no use worrying about it whatsoever. But eventually to satisfy the patient I had a Wassermann done, and it was frankly positive. Then the moral follows. These patients, having received adequate treatment for syphilis, have now and have had for a considerable period negative Wassermanns, but continue to have their functional nervous disorder as before.

Dr. W. F. Lorenz, Mendota, Wis.: For the last year and a half we have been making routine Wassermann tests on every admission to the three state hospitals of Wisconsin. Thus far over 3,500 cases have been examined. Among these not infrequently we found cases of manic depressive insanity with a positive Wassermann reaction. It has been the practice to make a spinal fluid examination in every case where the blood serum is positive. A number of cases were thereupon shown to be early paretics. On the other hand, the occurrence of syphilis among manic depressive cases, in which the syphilis is simply incidental, has been in the neighborhood of 5 per cent. The failures to recognize paresis have been in cases which simulate the manic phase of manic depressive insanity. A few years ago I reported a case of manic depressive insanity in which syphilis was contracted after three hospital admissions had occurred. Eight years after the infection, the case was admitted as a typical paretic with positive blood serum and spinal fluid. The clinical picture of paresis was not affected by the constitutional manic depressive insanity. I would

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like to emphasize the fact that paretics may very frequently simulate acute mania, and that routine Wassermanns and spinal fluid examinations may be the only means of diagnosing the condition.

Dr. Milton Board, Louisville, Ky.: As I understand the manic depressive group, we have to deal absolutely with hereditary insanity. I have never seen a case of manic depressive insanity in which I could not get, by careful research among the members of the immediate family, a distinct family history. I know of no reason why an individual with such a line of heredity should be exempt from contracting syphilis. He can have syphilis like any one else. And, having syphilis, and showing manifestations which we classify as belonging to the manic group, naturally you will find in the blood and in the spinal fluid the reaction of a syphilitic infection. You will get improvement as far as the syphilis is concerned under specific treatment. You will get improvement from the mental symptoms because in the ordinary run of those cases they tend to recover. The two conditions are entirely separate and distinct, and yet they can and do occur in the same individual. Furthermore, it occurs to me and it is proved that an individual may belong to this type of neurotic individuals which causes him to be susceptible to and present these mental symptoms which we classify as manic depressive insanity, and yet same individual may contract syphilis and later on after one, two, or three attacks of manic depressive insanity, with the recovery that we expect, true paresis may ensure. With our knowledge of the pathology of paresis, however, we know that we then have to deal with a condition which is progressive, which is not going to recover under any treatment that we now have or that, in my judgment, we will ever have, because in true paresis we are dealing with nerve structure which is already dead, and we are as powerless to cure that condition as we are to raise a dead body.

Dr. Julius Grinker, Chicago: Among other things Dr. Barrett's paper emphasizes the fact that one should not overestimate the importance of a positive Wassermann finding, as has become the custom quite recently. A positive Wassermann test does not necessarily mean that the patient is suffering from active syphilis, nor does it indicate that the condition he is suffering from is directly or indirectly the result of syphilis or parasyphilis. Mistaken diagnoses are often made because of the fact that the Wassermann test is positive, while the patient is at the same time suffering from an alcoholic or other psychosis. In my opinion the diagnosis of general paresis should be made only when the neurologic signs are present. Especial attention should be paid to the pupillary reactions, deep reflexes, and speech disturbances, and last but not least, to the positive

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Wassermann findings in both blood and spinal fluid. The mere finding of a positive Wassermann without the neurologic signs of general paresis does not, I believe, justify a diagnosis of general paresis. On the other hand I have seen cases of manic depressive insanity in which there was a positive Wassermann, and which were falsely diagnosed as general paresis. Subsequently they recovered from the manic depressive symptoms, and, after treatment also from the symptoms of syphilis.

DR. C. EUGENE RIGGS, St. Paul: About four or five years ago I saw a case in which the Wassermann was positive, both in the blood and spinal fluid. There was lymphocytosis and globulin excess. I thought the case was one of general paresis. There was absence, however, of the characteristic neurologic findings to which Dr. Grinker has referred. Since this patient made a satisfactory recovery, it was evidently a manic-depressive case.

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SPINAL FLUID FINDINGS CHARACTERISTIC OF CORD COMPRESSION *

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On two consecutive days of October, 1915, our attention was forcibly called to the significance of certain changes in the spinal fluid characteristic of cord compression. Together we performed a necropsy on a man in whom the diagnosis was obscure, as shown by the term "myelitis," which had been the most satisfactory one obtainable. His spinal fluid had been examined by one of us a week previously; it was yellow, contained a fibrin clot, showed great excess of proteins by ammonium sulphate and alcohol precipitation, but only one cell could be found and the Wassermann and culture was negative. Necropsy showed compression of the cord by an extradural abscess. The very next day a patient came to operation because of increasing symptoms of transverse myelitis; he had previously had yellow fluid, increased proteins, increased cells and positive Wassermann; in him a dense fibrous thickening of the pia enveloping the spinal cord was found, sufficient cause for the symptoms of transverse myelitis.

On talking with colleagues at our own hospital, we found that a fluid characteristic of cord compression was not recognized by them; nor did we fare better on questioning physicians and surgeons of other Boston hospitals. Fearing local ignorance, a number of men

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in nearby cities most likely to see such fluids were approached; some had seen one or two yellow fluids which had greatly puzzled them, others had heard of them and not seen them.

On turning our attention to the literature, however, we were promptly rewarded by finding a number of articles, especially in French and German, dealing with this subject, and in these countries it was evidently recognized that there are certain changes in the spinal fluid characteristic of cord compression, colloquially referred to as the "spinal fluid compression syndrome." We have been able to find no collection of a series of cases illustrative of this subject in American journals, although a number of isolated case reports have been presented, occasionally with excellent discussion of the literature, particularly the articles by Mix.[1]

A group of twelve cases is here presented, all personally known to one or both of the writers, either from the laboratory or the clinical side, and usually from both points of view. All show the spinal fluid compression syndrome in its complete or incomplete form, and in all we have proof of cord compression, either from operation or necropsy, in one case only resting our proof on a combination of clinical and Roentgen-ray evidence. It has been hard indeed to exclude five other cases of similar nature, in which proof of the lesion or lack of personal knowledge of the case was lacking; suffice it to say, that this rejected group is in perfect accord with the twelve which are presented. Before proceeding to our own cases, however, let us examine the literature.

REVIEW OF LITERATURE

Abnormal findings in the spinal fluid, which have now become regarded as indicative of pressure on the spinal cord, were observed by Froin[2] in 1903. The spinal fluids from his cases showed three abnormal characteristics, a yellow color or xanthochromia, spon-

1. Mix: Clinics of John B. Murphy, 1915, iv, 317.
2. Froin: Gaz. d. hôp., 1903, lxxvi, 1005.

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taneous coagulation, and an increase in the cell count. Three cases were reported at first and later one more.[3] Although no operative or postmortem findings are given, the histories of these patients would lead us to believe that the first was probably a pachymeningomyelitis of the conus terminalis, the second (?) tuberculous meningitis, the third Landry's paralysis, and the fourth syphilitic meningitis. From 1903 on, cases with these findings in the spinal fluid have been reported almost every year and Mix[1] in 1915, after a careful review of the literature, was able to find thirty-three and to add one case of his own. Since that time cases showing this complete syndrome have been reported by Demole[4] and Bromer.[5]

These thirty-six cases fall into three groups (1) the meningitis cases, (2) the cases showing pachymeningomyelitis of the conus terminalis, and (3) the cases of cord tumors. Under the first heading, meningitis cases, are found two of Froin's cases, one reported by Sicard and Descomps,[6] one of Froin and Foy's,[7] one by Verhoogen and Dustin,[8] one by Mestrezat,[9] one by Marinesco and Radovici,[10] one by Debré and Paraf,[11] and one by Bromer. Cases of Landry's paralysis reported by Froin, Donath,[12] and Braillon[13] also come into this first group. A typical example of this meningitis group is the case reported by Mestrezat:[14] Clinically there was flaccid paresis of the lower extremities of sudden onset with pain, loss of sphincter control, and with sensory and trophic changes. The spinal fluid one month after onset showed massive coagulation and xanthochromia and many lymphocytes. Necropsy showed

3. Froin: Aubry's Thèse de Paris, 1909.
4. Demole: Rev. neurol., 1915, xxviii, 648.
5. Bromer: Am. Jour. Med. Sc., 1916, cli, 378.
6. Sicard and Descomps: Gaz. d. hôp., 1908, lxxxi, 1431.
7. Froin and Foy: Gaz. d. hôp., 1908, lxxxi, 1587.
8. Verhoogen and Dustin: Soc. d. sc. méd. et nat. d. Brux., 1909, lxvii, 169.
9. Mestrezat: Liquide céphalo-rachidien normal et pathologique, Paris, 1912.
10. Marinesco and Radovici: Rev. neurol., 1913, xxv, 268.
11. Debré and Paraf: Presse méd., 1913, xxi, 952.
12. Donath: Wien. klin. Wchnschr., 1905, xviii, 1327.
13. Braillon, Gaz. d. hôp., 1909, lxxxii, 635.
14. Mestrezat: Footnote 9, p. 432.

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extradural inflammatory neoformation at the level of the eighth and the ninth dorsal segments cutting off the end of the cord, with extension of inflammatory process to the leptomeninx.

The second group of cases is that best classified as pachymeningomyelitis of the conus terminalis, cases such as have been reported by Froin, Babinski,[15] Cestan and Revaut,[16] Fornaca,[17] Tedeschi,[18] Derrien, Mestrezat and Roger,[19] Claude,[20] Mestrezat, Marinesco and Radovici, and Launois, Frolin and Ledoux.[21] Some of these cases were without operation or necropsy, so that exact classification is impossible. One illustration of this group may be cited. The case of Launois, Froin and Ledoux is as follows: At first there was monoplegia, later flaccid paralysis, incontinence, Babinski, increasing paraplegia and death. The duration of the symptoms was four months. The spinal fluid formed a spontaneous clot with xanthochromia and lymphocytes. Necropsy showed chronic pachymeningtis with cutting off of dural cone at level of the conus terminalis.

The third group of the tumor cases all showed xanthochromia and spontaneous coagulation, but a variation in the cell count. Two examples of this group are as follows: In a case of Blanchetiere and Lejonne,[22] a man of 66 had clinically spastic paraplegia. The spinal fluid finding showed yellow fluid, coagulation en masse, and few lymphocytes, found on seven punctures. Postmortem examination showed a tumor of the dura mater on the dorsal surface, at the seventh, eighth and ninth thoracic segments, not adherent to the cord. There was distinct compression of the cord, but no signs of meningitis. The duration of the symptoms before puncture was four months. A case

15. Babinski: Presse méd., 1903, xi, 755.
16. Cestan and Ravaut: Gaz. d. hôp., 1904, lxxvii, 985.
17. Fornaca: Gazz. d. osp., 1906, xxvii, 497.
18. Tedeschi: Gazz. d. osp., 1906, xxvii, 1067.
19. Derrien, Mestrezat and Roger: Rev. neurol., 1909, xvii, 1077.
20. Claude: Rev. neurol., 1909, xvii, 1420.
21. Launois, Froin and Ledoux: Gaz. d. hôp., 1914, lxxxvii, 361.
22. Blanchetiere and Lejonne: Gaz. d. hôp., 1909, lxxxii, 1303.

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reported by Klieneberger[23] was clinically a case of flaccid paraplegia. The spinal fluid findings showed increased pressure, amber color, spontaneous clot, increase in lymphocytes, on two punctures. On operation there was found a tumor of the fibers of the cauda equina and the anterior wall at the level of the second and the third lumbar vertebrae. A clear yellow fluid under pressure was seen below the tumor. The duration of the symptoms was about six months. Other cases in this group have been reported by Flatau,[24] Rindfleisch,[25] Collins and Elsberg[26] and Mix. All of these cases show xanthochromia and spontaneous coagulation.

Unlike the cases of meningitis there was only a slight cell count or none at all. This last fact was specially noted by Blanchetiere and Lejonne, who criticized the observation of Secard and Descamps, that all such spinal fluids were due to meningomyelitis. Mix also notes that the inflammatory compressions are the ones that give high cell counts. Thus he concludes that this characteristic is not a necessary part of the syndrome.

In regard to the cause of these findings there has been marked differences of opinion. Froin made no attempt to generalize. Cestan and Ravaut were the first to attempt to explain the xanthochromia and coagulation. They thought that the deep yellow color was due to the dissolution of the red blood corpuscles and consequent freeing of hemoglobin. The increase in albumin was due to the serofibrinous exudate of the inflammation. They even went so far as to think that a diagnosis of fibrinous hemorrhagic meningitis could be made from the spinal fluid findings. Sicard and Descomps believed that the inflammation was annular and caused vascular compression and the formation

23. Klieneberger: Monatschr. f. Psychiat. u. Neurol., 1910, xxviii, 346.
24. Flatau: Sektion d. neurol.-psychiat. Gesellsch z. Warschau, Nov. 19, 1910; Ztschr. f. d. ges. Neurol. u. Psychiat., 1911, iii, 146; Rev. neurol., 1911, xxi, 440.
25. Rindfleisch: Deutsch. Ztschr. f. Nervenh., 1904, xxvi, 135.
26. Collins and Elsberg: Am. Jour. Med. Sc., 1914, cxlvii, 493.

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of a meningeal pouch below the point of constriction. As no tumor case had been reported up to that time they believed that the syndrome was always diagnostic of meningitis.

With the publication of the first tumor case by Blanchtiere and Lejonne in 1909 the early theory of meningomyelitis as being the only cause had to be abandoned. These workers thought that the xanthochromia was derived from the hemoglobin. Kleineberger in 1910 believed that the tumors in his cases divided the spinal canal into two parts. Launois, Froin and Ledoux in 1914 came to the conclusion that the yellow color was due to local hemolysis or an exudation of the blood plasma. Mestrezat[27] concludes: "that it is a transudation and perhaps sometimes little capillary hemorrhages in a closed pouch in the lumbar culdesac isolated in different ways from the remaining subarachnoid space, which brings about the syndrome of massive coagulation and xanthochromia."

It was soon noted that many cases of obvious spinal cord pressure due especially to tumors did not give a spinal fluid showing the complete syndrome of xanthochromia and coagulation en masse, with fibrin clot, but did show a marked increase in protein content, entirely out of proportion to the number of cells present. Nonne[28] in 1910 seems to have been the first to note this point and in that year he published a report of six cord tumors with spinal fluids of a pathologic nature. Nonne's cases did not show spontaneous coagulation as had the carefully reported case of Blanchetiere and Lejonne's, but all showed a very strong Phase 1 reaction with few or no cells. Three cases were verified by operation and two by postmortem examination.

The next important paper was one by Raven[29] from Nonne's clinic in 1912. Raven gives a careful review of the literature and reports fifteen cases, each showing an increase in proteins, and some showing spon-

27. Mestrezat: Footnote 9, p. 448.
28. Nonne: Deutsch. Ztschr. f. Nervenh., 1910, xl, 161.
29. Raven: Deutsch. Ztschr. f. Nervenh., 1912, xliv, 380.

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taneous coagulation and xanthochromia. All these cases came to operation or necropsy and all showed a definite lesion causing compression of the spinal cord, the large majority being tumors. Raven also gives a table of forty-seven cases found in the literature, including his own fifteen and the six previously reported by Nonne. The focus of compression varied, eight being cervical, eighteen dorsolumbar, eight conus or cauda, and five diffuse. His conclusions are as follows:

Albumin increase with slight or no lymphocytosis indicates compression of the spinal cord. The cause of the compression can not be determined by this test. The increase of albumin occurs through stasis of fluid below the site of compression. Xanthochromia results from hemoglobin. Its presence strengthens the diagnosis. The syndrome may be found in cases of intramedullary tumors. The article is illustrated by two good colored reproductions of spinal fluids showing xanthochromia.

Later Raven[30] adds seven more cases to the fifteen already reported by him. In this paper he said that he was trying to find out the point of compression and the cause of compression, but this he was unable to do at that time. He reiterated his belief that a spinal fluid showing increased proteins and xanthochromia means compression at some point on the spinal cord. Nonne[31] in 1913 reported sixteen more cases, giving in detail the history of each patient with operative and postmortem findings and many exceptionally good photographs of the lesions. Many are tumor cases and the majority show increased proteins and some xanthochromia in the spinal fluids. Two cases are specially notable: His Case 3, gumma of the meninges in the dorsal region pressing on the spinal cord; the first lumbar puncture showed lemon color, Phase 1, triple plus, no lymphocytes and a positive Wassermann

30. Raven: Deutsch. Ztschr. f. Nervenh., 1913, xlix, 36.
31. Nonne: Deutsch. Ztschr. f. Nervenh., 1913, xlvii, 436.

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in both blood and spinal fluid. After intensive antisyphilitic treatment with clinical improvement the spinal fluid became clear and colorless. Phase 1, plus showed no lymphocytes and Wassermann negative. In speaking of the value of lumbar punctures as diagnostic measures, Nonne says that these two punctures showed that there was compression of the spinal cord, that it was due to syphilis, and that the second puncture gave ocular demonstration of the improvement under treatment. Case 5 was osteoma of the tenth dorsal vertebra causing marked extradural compression. Clinically it was transverse myelitis. The spinal fluid was clear, Phase 1, triple plus, cells eight, Wassermann negative in both blood and spinal fluid. Three good illustrations of the cord are shown.

The following conclusions are drawn by Nonne:

1. The examination of spinal fluids seems to give us a compression syndrome by the increase in globulin with slight or no lymphocytosis, and either with or without a yellow color.

2. The increase of albumin in the spinal fluids in cases of tumor is probably due to stasis distal to the point of compression and not to inflammation.

The following diagnostic points are given:

1. Isolated increase of albumin in the fluid strengthens the suspicion of compression of the cord.

2. It is present only distal to the point of compression.

3. Xanthochromia of the fluid is due to mixture with blood coloring matter, probably in consequence of hemorrhage.

4. The syndrome gives no inference as to the character or starting point of the compression.

5. Spinal fluid results do not differentiate intramedullary and extramedullary growths.

Rare, isolated cases are found in the American and foreign literature, often reported without special attention to the spinal fluid findings. Such cases are

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those of Bassoe,[32] Schaller,[33] Assmann,[34] and Skoog.[35] Demole[4] in 1915 reported a case of carcinoma of the vertebrae metastatic from the breast, which showed massive coagulation and xanthochromia in the spinal fluid. Reichmann[36] in 1912 reported a case of von Rechlinghausen's disease with multiple metastases in the spinal canal giving the syndrome of xanthochromia and increased proteins in the spinal fluid. Bromer[5] reported in 1916 a case of Pott's disease in the cervical region, the spinal fluid showing spontaneous coagulation and xanthochromia.

In summarizing the literature the following points may be mentioned:

1. Abnormal findings in the cerebrospinal fluid in cases of cord compression was first noted by Froin in 1903. Since that date over fifty cases have been reported, mostly in the French and German literature.

2. The original syndrome of Froin consisted of xanthochromia, spontaneous coagulation with increase in the cell count. Other workers have shown that compression is also indicated by a low cell count, marked increase in protein content, with or without xanthochromia (Nonne and Raven).

3. The cause of compression may be tumors, Pott's disease, circumscribed meningitis, meningomyelitis, or any pathologic process causing complete or partial transverse myelitis from pressure, within or outside of the cord.

4. Compression may be exerted at any point on the spinal cord, but in the majority of the cases reported it was in the lumbar region or the conus terminalis.

5. The pathologic fluid was found only distal to the point of compression.

6. The cause of these findings is not known. It seems certain that in all of the cases reported there

32. Bassoe, J.: Nervous and Mental Diseases, 1915, xlii, 736.
33. Schaller: J. Nerv. and Ment. Dis., 1913, xl, 489.
34. Assmann: Deutsch. Ztschr. f. Nervenh., 1910, xl, 131.
35. Skoog: Spinal Cord Compression from Leptomeningeal Cysts, THE JOURNAL A. M. A., July 31, 1915, p. 394.
36. Reichmann: Deutsch. Ztschr. f. Nervenh., 1912, xliv, 95.

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was the formation of a culdesac below the point of compression and it is in this walled-off space that the abnormal spinal fluid is found.

ANALYSIS OF PERSONAL CASES

1. *Characteristics of the Spinal Fluid.*—The one outstanding feature of the fluid in these cases is the marked increase of protein content. Normally, the spinal fluid contains a trace of proteins, as may be demonstrated by precipitation with alcohol; the fluid in cases of chronic inflammation of the meninges, such as tabes, shows slight increase, as demonstrated by alcohol, ammonium sulphate (Nonne), butyric acid (Noguchi) and other tests. The increase in these cases is usually many times that of the tabetic fluid.

That this increase of proteins is not dependent on an inflammatory process is evident from the fact that cells are either absent or in negligible quantity, and when found, are frequently endothelial in origin and not exudative in type; in two cases, in which a definite lymphocytosis occurred, the constrictive process was shown to be syphilitic. Interesting in this connection is Case 8, in which we have spinal fluid examinations before and after the appearance of symptoms of compression; with no more evidence of inflammation we see a marked increase in the protein content.

Xanthochromia occurred in just half of our cases, the color varying from the palest yellow tint to a deep yellow. The remaining six cases showed clear, colorless fluids, to the eye apparently normal. In one patient, in whom consecutive punctures were made, we were able to follow the fluid in its transition from colorless to yellow and to colorless again after operation. Whether colorless or xanthochromic, the fluids were invariably clear, with the exception of fibrin clots, which formed spontaneously in four of the most deeply yellow fluids.

Pressure, as tested by manometer, was invariably within normal limits, tending toward low readings. An additional argument that there exists a closed cav-

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ity below the level of constriction is given in Case 6; this patient had brain tumor with beginning choked disk and should have had high spinal fluid pressure, 400 mm. or more; his pressure was normal, 170 mm. It is reasonable to assume that the tumor, which also constricted the cord, had shut off the lower from the upper spinal canal.

The Wassermann reaction was negative in all except three cases, two of these undoubtedly syphilitic.

The colloidal gold test of Lange was performed in eleven cases. In seven the greatest color change appeared in the maximum dilutions, the so-called tumor zone. In one it was in the syphilitic zone, and in three there was a slight color change in all dilutions. In two patients in whom goldsol reactions were performed at different times the maximum change was seen to occur in progressively greater dilutions as the clinical picture of transverse myelitis became more marked, this change going hand in hand with increase in proteins.

Culture was negative in one case of epidural abscess, thus showing what an important barrier the dura presents to infection by direct extension.

To sum up, all fluids showed increased proteins, all except one greatly increased. There was no evidence of acute meningeal irritation in any and there was evidence of slight chronic irritation, as shown by mild lymphocytosis, in two only. Half of our cases showed the yellow color and four of these exhibited the full characteristics of the original *syndrome de coagulation massive et de xanthochromie* of the French. The remaining six fluids are, however, pathologic only to a less extent, and are characteristic of cord compression, though less strikingly so. Pressure of spinal fluid was essentially normal in all tested cases. The Wassermann reaction was strongly positive only in syphilitic cases. Goldsol reaction usually fell in the tumor zone, with marked change of color in the greater dilutions.

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TABLE 1.—DATA OF AUTHORS' CASES OF CORD COMPRESSION, ARRANGED IN SUCCESSION FROM HEAD DOWN

Case No.	Spinal Fluid Syndrome	Compressing Agent	Location and Aspect of Compression	Duration of Compression	Intensity of Symptoms
10	Incomplete	Dermoid cyst of cord	Cervical 4 and 5, central pressure	6 years	+++
3	Incomplete	Tuberculosis of vertebra	Cervical 6 and 7, ventral pressure	4 weeks	++
5	Complete	Intramedullary tumor	Cervical 7, central pressure	7 years	++
9	Complete	Chronic progressive fibrous leptomeningitis (syphilitic)	Diffuse; all sides upper dorsal	1 month	+++
7	Incomplete	Intramedullary tumor?	Dorsal 4, central pressure	13 years	+
1	Complete	Epidural abscess	Dorsal 4 and 5, dorsal pressure	1 week	+++
6	Complete	Glioma of meninges	Diffuse, all sides, mostly dorsal	4 months	+++
2	Incomplete	Extradural endothelioma	Dorsal 6, dorsal pressure	1½ years	++
11	Complete	Epidural abscess	Dorsal 4 to 7, dorsal pressure	2 weeks	+++
8	Incomplete	Charcot spine	Dorsal 11 and 12, lumbar 1 and 2	14 weeks	+
12	Complete	Varix of cord	Sacral, central pressure	5 years	++
4	Incomplete	Fibroma	Cauda equina	4 to 6 years	+

TABLE 2.—SYMPTOMS, FINDINGS, TREATMENT AND RESULT IN AUTHORS' CASES OF CORD COMPRESSION

Case Number and Condition Present	Symptoms and Signs	Spinal Fluid	Result
1. Pressure on cord from extradural abscess	Complete transverse myelitis, middorsal; one week	Yellow, clear, with fibrin clot; coagulable proteins greatly increased; cells 0; pressure normal; culture negative; goldsol meningitis	Necropsy. Pressure on D. 4 and 5 segments, posterior and lateral, by pus, extradural
2. Progressive paralysis from extradural endothelioma	January, 1914. Brown-Sequard syndrome 8 weeks' duration. Probable diagnosis, syringomyelia	Colorless, clear; proteins much increased; cells 8; Wassermann negative; goldsol: 0, 0, 1-2, 2, 2, 3, 2, 2, 1, 0	
	December, 1914. Increase of symptoms, approaching transverse myelitis. X-ray of spine negative	Colorless; proteins much increased; cells 1; Wassermann negative; pressure 150 mm.; goldsol: ±, 1, 2, 2, 2-3, 2-3, 1, ±, 0, 0	
	August, 1915. Partial transverse myelitis at D. 4. Unable to walk. Double clonus and Babinski, more marked on right	Colorless; proteins much increased; cells 5; Wassermann negative; pressure 210 mm.; goldsol: 0, 0, 0, ±, 1, 1-2, 1-2, 1, ±, 0	Aug. 10, 1915. Operation. Endothelioma extradural, size of English walnut pressing on D. 6 and attached to dura on right. Erosion of lateral wall of vertebral canal into base of 4th D. spine. Tumor removed entire, dura incised, with escape of clear fluid. Complete recovery of patient

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TABLE 2.—SYMPTOMS, FINDINGS, TREATMENT AND RESULT IN AUTHORS' CASES OF CORD COMPRESSION—Continued.

Case Number and Condition Present	Symptoms and Signs	Spinal Fluid	Result
3. Pressure from Pott's disease	July, 1914. Partial transverse myelitis, 4 weeks. X-ray shows distinct difference in articulations of lower cervical vertebrae	Colorless; proteins much icreased; cells 26; Wassermann negative; goldsol: 0, 0, 0, ±, 1, 3, 3, 5, 4, 2; culture negative	Operation. Cord bulging of anterior wall of spinal canal caused by extradural tubercular abscess. Improved
4. Fibroma of cauda equina	September, 1915. Pain and paralysis of legs, without loss of sphincters or sensory disturbance characteristic of transverse myelitis; slowly progressive	Colorless; proteins much increased; cells 40; Wassermann negative; goldsol: 0, 0, 0, ±, 2, 3, 3-4, 3, 2, 1; pressure 110 mm.	First operation, no tumor found (too low). Second operation, fibroma found on one part of cauda equina, molded to the shape of spinal canal
5. Intramedullary tumor	Illness began 7 years ago with pain in left arm which became, 4 years ago, replaced by numbness; later, stiffness of legs. X-ray showed distinct atrophy of 7th cervical vertebra	Yellow; proteins much increased; Wassermann negative; goldsol: 0, 0, ±, 1, 1, ±, 1, 2, 3, 3; pressure normal	Operation. Swollen, discolored cord at C. 7 level. Diagnosis, intramedullary tumor. Partial benefit
6. Glioma of brain; extension to spinal meninges	Marked ataxia of legs of few weeks' duration, rapidly progressive to motor and sensory paralysis with upper level at D. 8. Optic neuritis, developing into choked disk, and symptoms of increased intracraniall pressure. X-ray of spine and cranium negative	Oct. 21, 1915. Yellow; no clot on standing; proteins much increased; cells 11; Wassermann negative; goldsol: 1, 1, 1, 1, 1, 1, 1, 1, 1, 1; pressure 170 mm. Nov. 17, 1915. Slightly yellow; cells 4; goldsol: 0, 0, 0, 0, 0, ±, 1, 1, ±, ±	Nov. 9, 1915. Operation. Subtemporal decompression; no tumor found Nov. 17, 1915. Necropsy. Glioma at base of brain, and also extending up and down cord for greater part of its length, between membranes, most marked and entirely surrounding cord in dorsal region
7 Intramedullary disease (?)	For 13 years increasing paresthesiae of fingers and later feet. Recently cutaneous sensory disturbance to level of upper dorsal. X-ray negative	Colorless; proteins moderately increased; cells 0; pressure normal; goldsol: 0, 0, ±, ±, ±, 0, 0, 0, 0, 0; (Wassermann blood and fluid negative previously)	Operation. Discolored cord at D. 4. Cord not enlarged
8. Charcot spine	1913. Typical low tabes of 2 years' duration. Treated with salvarsan and mercury, without change	Colorless; proteins slightly increased; cells 78; pressure 60 mm.; Wassermann negative (later positive); (Wassermann blood positive)	
	1915. Cutaneous sensory disturbance with Babinski. Knuckle of lumbar spine noted for first time. X-ray showed fusiform swelling about and involving high lumbar vertebrae	Colorless; proteins greatly increased; cells 21; Pressure 100 mm.; Wassermann strongly positive; goldsol: 0, 0, ±, 2, 2, 2, 3, 1-2, ±, 0	Plaster cast gave partial relief

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9 Chronic progressive syphilitic leptomeningitis	1908. Treated for syphilis of nose. 1913. Left 6th nerve palsy, 6 months' duration, with pains in legs and unsteadiness. Argyll Robertson pupils, but normal reflexes. Improved under salvarsan	Colorless; cells 10; Wassermann negative; (proteins not examined)	
	March, 1914. Better.....	Colorless; cells 44; Wassermann positive; pressure 160 mm.; (proteins not examined)	
	August, 1914. Complains of pain about shoulders and stiffness in walking	Yellow, clear; proteins much increased; cells 1; Wassermann anticomplementary; pressure 110 mm.	
	Sept. 21, 1914.....	Yellow, clear; proteins much increased; cells 1; pressure 100 mm.; Wassermann anticomplementary; goldsol: 0, 0, 4, 4, 4-5, 5, 5, 5, 5, 5	
	Oct. 1, 1914.....	Slightly yellow, clear; proteins much increased; cells 11; pressure 170 mm.; Wassermann anticomplementary; goldsol: 0, 1, 2, 2, 2-3, 3, 3-4, 3-4, 3, 1	
	Nov. 7, 1914. No change in condition clinically. Clearly spastic paraplegia with diminished cutaneous sensation below nipples	Yellow, clear; proteins much increased; cells 5; pressure 150 mm.; goldsol: 0, ±, 1, 2, 2, 2-3, 3, 3, 2, 1	
	December, 1914. Same. Babinski and clonus both sides. Atrophy shoulder girdles		
	July 16, 1915. Condition same.....	Colorless; proteins moderately increased; cells 5; pressure 90 mm.; goldsol: 0, 0, 0, ±, 1, 1, 1, ±, 0, 0	Oct. 8, 1915. Operation. Extensive dense fibrous leptomeningitis encasing cord posteriorly and laterally in upper dorsal region, extending above and below laminectomy opening. Incised but cannot be removed
	Oct. 23, 1915. Condition about same.....	Bright yellow; proteins much increased; cells 3; pressure 170 mm.; goldsol: 0, 0, 0, ±, ±, ±, 1, 2, 2, ±	
	Feb. 10, 1916. Same. Perhaps a little improvement in motion and sensation	Colorless, clear; proteins moderately increased; cells 4; pressure 100 mm.; goldsol. 0, 0, 0, ±, 1, 1-2, 3, 3, 3, 2-3	
10. Large dermoid cyst of cord	March, 1916. Pain in arms for 6 years. For one year weakness of legs and numbness of arms and legs; marked spastic gait with crutches. Cutaneous sensory change to C. 4	Colorless, clear; proteins slightly increased; cells 1; Wassermann negative; goldsol: 0, 0, 0, ±, 1, 1, ±, 0, 0, 0	Operation. Dermoid cyst, apparently arising from and markedly distorting the spinal cord at C. 4 and 5. May 3, able to stand and walk a little better
11. Pressure on cord from extradural abscess	Feb. 18, 1916. Pain in left shoulder and chest 2 weeks. Retenton 5 days. Spastic paraplegia 4 days. Temp. 101 to 105. Hyperesthesia. D. 7 to 9. Anesthesia below D. 9	Yellow, clear; fibrin clot; protein greatly increased; cells 0	Operation. Epidural abscess pressing on cord, D. 4 to 7. Culture from pus shows Staphylococcus aureus. Death; no necropsy
12. Varix of posterior spinal veins of sacral cord	Pain in spine, back and legs 5 years. Disturbance sphincters 1½ years. April 25, 1916. Knee jerk, left greater than right. Ankle jerk not obtained. Plantar reflexes normal. Atrophy right leg with toe drop. Sensation normal	Bright yellow, clear; fibrin clot while needle in situ; protein much increased; pressure not increased; cells 4; Wassermann negative; goldsol: 0, 0, 0, ±, 1, 1-2, 3, 3-4, 4, 5	Operation, May 1, 1916. Cord seemed to be under pressure and forced outward on opening dura. Veins of sacral cord greatly dilated at first, but normal after few minutes. No other abnormality. Patient improved

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2. *Constricting Agent.*—Even in this small group we find many different types of causes and degrees of constriction. We have agents causing compression for a week and for years; we have pressure caused from outside of the dura, from inside of the cord and from affection of the meninges themselves. We have acute inflammations and chronic inflammations; we have rapidly growing tumors and benign tumors.

Again, we find that our series shows constriction of nearly every segment of the spinal cord, and even a tumor below the cord is found to give the characteristic spinal fluid picture. By far the greater number of cases fall in the dorsal region, as is to be expected from its relatively greater length and variety of pathologic processes involved.

Concerning the aspect of the cord on which compression is chiefly exerted, we find pressure from the side, from back, from front, constriction from all sides and pressure from within; in fact, every aspect imaginable.

3. *Probable Duration of Compression.*—In these cases this was as varied as the pathologic process and the location and aspect of the compressing agent. For the most part, however, they fall into two groups: compression from acute processes, such as extradural abscess leading to rapid paralysis, and compression from chronic processes, causing insidious symptoms. All, however, produced spinal fluids which gave the syndrome, complete or incomplete.

A glance at Table 1 will give a concise idea of the above facts, and will help to understand the subsequent discussion. Intensity of symptoms is shown by a plus sign, depending on the degree of disability, both motor and sensory, exhibited at the time of puncture. Spinal fluids are classed as forming the complete or incomplete syndrome, depending on whether they exhibit xanthochromia or not. The arrangement is topographic from cervical to sacral downward.

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COMMENT

Can we go farther than to differentiate two general varieties of spinal fluid indicative of cord compression? Can we deduce anything of more minute diagnostic value from the study of even these few cases?

It seems to us that we can. One point seems certain, pressure sufficient to cause marked sensory and motor disturbance tends to give the complete spinal fluid syndrome. The one exception was Case 10, in which the locus of pressure was the highest in the series; and this leads to the second point, that in cases where symptoms of compression were marked, the lower loci of compression more frequently gave the stronger positive fluids. Compression from acute processes was more potent in the production of yellow fluids than that from chronic causes, and, lastly, it seems that intramedullary and meningitic lesions, especially when surrounding the cord completely, are more productive of the complete syndrome than are extradural processes.

It seems reasonable at this point to take into consideration five other cases which have come to our attention, which could not, for lack of proof, be used before; all are clinically cord pressure cases, and one proved by necropsy (case of Dr. Charles A. McDonald of Providence); four of these five had punctures from forty-eight hours to six weeks after onset of symptoms; all gave the complete spinal fluid picture. The fifth case shows little disability, but symptoms are referable to the lumbar cord; the fluid in this case also is yellow.

In line with our finding of incomplete fluids as a result of extradural pressure from chronic processes is the only case which we have had which failed absolutely to give positive findings. This patient gave the clinical picture of transverse myelitis in moderately complete form, with level at dorsal 5 to 7. Operation revealed myeloma of the vertebra, with extradural pressure on the cord. This is in agreement with

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Nonne, who found negative fluids in a few cases with evident cord pressure. On the other hand, in upward of 1,000 punctures we have not found the syndrome in other than cases of cord compression. While, then, we must admit that spinal cord compression may be present without causing changes in the spinal fluid below the pressure level, the fact seems to be that the characteristic changes usually occur, and that we can reasonably expect to find the spinal fluid syndrome in its complete form in cases in which symptoms of pressure develop rapidly, especially if the compressing agent is within the dura and the pressure is exerted on the lower part of the spinal cord.

CONCLUSIONS

1. We believe that changes in the spinal fluid frequently occur as a result of compression of the spinal cord.

2. The principal characteristic of such compression fluid is marked increase of proteins without corresponding cellular increase, obtained under normal pressure.

3. Xanthochromia with massive coagulation added to the above, makes a more intense reaction of the same significance, only more conclusive.

4. Factors which tend to give the syndrome most readily, or to give it in its most intense form, are processes which act rapidly, are intramedullary or cause pressure on the cord from all sides, and which affect the lower cord.

5. We do not believe that such findings are necessary in cord compression, but that their presence is confirmatory evidence of value in some cases.

We wish to express our thanks to Dr. E. W. Taylor, chief of clinic, and to Dr. W. J. Mixter, who operated on a number of the patients, for their assistance in the preparation of this paper.

ABSTRACT OF DISCUSSION

DR. PETER BASSOE, Chicago: In an article by Dr. Charles L. Mix on massive coagulation and xanthochromia it is shown that this syndrome is not characteristic of either inflamma-

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tion or tumor, but of the formation of a meningeal pouch in the lower part of the spinal canal. Dr. Mix insists that only cases in which the fluid coagulates massively, so the tube can be turned upside down without spilling, should be included. In a recent case of sarcoma of the cord in the thoracic region I repeatedly obtained a uniformly yellow fluid which coagulated spontaneously but not massively.

Dr. Francis A. Ely, Des Moines, Ia.: Within the past six months I have had the opportunity of studying the cerebrospinal fluids in two cases. The first case was that of a young man who came into the hospital with symptoms which led to a provisional diagnosis of laminated cervical meningitis although there was marked loss of sensibility to pin prick and temperature discrimination. The predominance of tearing bilateral symmetrical pains in the distribution of the median and ulnar nerves and the marked loss of sensibility to cotton wool led me temporarily astray from the proper diagnosis. Fearing that I might be overlooking an intramedullary tumor the patient's consent to a cervical laminectomy was secured, and at operation the dura was observed to be nonpulsating and the cervical cord to be greatly swollen and distended. A subsequent necropsy revealed an extensively tubulated syringomyelic cord. The cerebrospinal fluid obtained by lumbar puncture when the patient first entered the hospital exhibited the xanthochromic syndrome and the fluid obtained from the cavity within the cord was found to have the same characteristics. The other case was one of posterior fossa tumor probably primary in the cerebellum and growing into the pontine angle, in which the cerebrospinal fluid also exhibited the yellow color, strongly positive Noguchi, low cell count, and negative Wassermann. In this case no evidence of compression of the cerebrospinal axis below the foramen magnum can be obtained.

Dr. A. L. Skoog, Kansas City: I have had similar experiences with cord compression cases due to several different causes. Perhaps the most interesting cases I have observed during the past ten months are two psammomas, both of them growing from the inner surface of the dura and both involving the mid-dorsal region. One had existed, judging from the clinical history, for a period of nine years, the other for about two years. Pathologically the tumors showed a difference owing to their age, the older one being much more dense and having a heavy capsule. The operation demonstrated that they had been localized correctly, and they were removed successfully. One of them showed a mild xanthochromia, the other not. Both of them had a greatly increased protein content in the spinal fluid. They were negative to Wassermann and gold colloid tests for syphilis. One interesting finding on which I lay a great deal of stress, and which I believe should be added to the syndrome, is the state of pressure of the cerebrospinal fluid. For instance,

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the first case cited had in the sitting posture a spinal fluid pressure of only about 40 mm., while the normal pressure is in the neighborhood of 120. After withdrawing, allowing it to drop away, not more than 3 c.c., the pressure was reduced to *nil*. The second case, again in the sitting posture, at first had a spinal fluid pressure of about 55 mm. (measured by the water-gage apparatus). After removing about 5 c.c. the pressure in this case also was reduced to *nil*. The first case had no lymphocytosis. The second patient, the one most recently operated on, had a lymphocyte count of 9.3 per cubic mm. In cerebrospinal syphilis there is practically always increased pressure. I know of but one exception in my experience, and at necropsy it was demonstrated that the choroid plexus was practically destroyed, which explained the subnormal spinal fluid pressure. I think that nine-tenths of the choroid plexus cells had been destroyed, but he had marked evidences of cerebrospinal lues.

Dr. Ernest Sachs, St. Louis: About two years ago I had a case of fibroma of the spinal cord, the patient having had symptoms for about four years. At the end of the first two years he was completely paralytic, had lost all power in the legs and had complete loss of sensation to the eighth dorsal segment. Following this he recovered the use of his legs to a slight extent. They became very spastic, and still he was bed-ridden. At the end of four years he came to me. The spinal fluid showed the characteristic picture of xanthochromia. It did not coagulate, and we made a diagnosis of spinal cord tumor at the level of the eighth dorsal segment. The two interesting points in the case were, first, that after withdrawing about 3 c.c. of yellow fluid, we were unable to obtain any more; second, that the tumor symptoms had receded, which was explained at operation by the fact that histologic examination of the tumor indicated that at one time it was very vascular but had contracted. As to the statement that xanthochromia is produced by complete compression of the cord, this case would seem to throw some doubt on that conclusion, since the tumor, having contracted, occupied less space in the spinal canal than had been the case originally. I would like to ask Dr. Ayer whether in any of his cases a second puncture yielded yellow fluid. In this case a second lumbar puncture showed no fluid whatever, and following operation the spinal puncture yielded normal cerebrospinal fluid. I think that the yellow color must be due to blood pigments. I do not see why a transudation should give a yellow color unless the color is imparted by some degeneration of the hemoglobin.

Dr. Charles R. Ball, St. Paul: In my experience xanthochromia is not confined to cord lesions, but in quite a number of cases that have come under my observation I have found it in lesions of the brain proper. This has been observed by me in a number of cases of severe apoplexy where two or

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three weeks afterward there was a very distinct yellowish tinge to the cerebrospinal fluid. Also in two cases of cerebrospinal syphilis there was very marked xanthochromia. These cases afterward cleared up under specific treatment. In the cerebral lesions, and in the cases of cerebrospinal lues, however, the cell count was quite large, so that the compression syndrome which Dr. Ayer describes in his paper was not present. In deciding on the diagnosis of spinal cord compression this syndrome to which Drs. Ayer and Viets have called attention is a very important one. I would have much more assurance in advising a decompression by laminectomy when it is present. It is well for us to keep in mind that xanthrochromia may be found in both brain and cord lesions.

Dr. W. F. Lorenz, Mendota, Wis.: I concur that xanthochromia does not necessarily indicate a case of spinal cord compression. In two cases that came to necropsy a focal lesion was found, and the condition was evidently the result of former hemorrhage.

Dr. Charles A. Elsberg, New York: Up to the present time we have been unable to discover anything characteristic in the spinal fluid in spinal cord tumor. In thirty cases of extramedullary spinal cord tumor that I have operated on there was xanthochromia in six, but five of these six cases were tumors of the conus and cauda equina, the other was a tumor in the lower dorsal cord. In a considerable number of my patients there was neither an increase of globulin nor an increase in the number of cells, nor anything else abnormal. In other words, in at least one-third of them the spinal cord fluid was absolutely normal in every respect. In the absence of abnormalities in the spinal fluid, therefore, there may nevertheless be pressure on the spinal cord. On the other hand, however, I have seen three cases of tumor near the cerebral ventricles, in which aspiration of the ventricle yielded a yellow fluid; in all three patients lumbar puncture also yielded a yellow fluid. Two of the cases came to necropsy, and were proven to be large tumors near the third ventricle. The third case, which did not come to necropsy, was operated, and the tumor was found. I do not believe, therefore, that the findings described by Dr. Ayer are absolutely characteristic of spinal cord tumor, although frequently present.

Dr. James Bourne Ayer, Boston: One of the speakers stated that xanthochromia occurred in other conditions, as tumors of the brain. I do not doubt it, but xanthochromia is not the syndrome. It is xanthochromia plus the other findings. We have had two cases showing yellow color, but with only very limited increase in proteids, one with many red blood corpuscles, the other with polynuclear leukocytes, both of which were unlike the fluids of cord compression. Dr. Elsberg is very skeptical in regard to the use of this syndrome. It is in differentiating certain other organic con-

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ditions affecting the spinal cord which may or may not be cord tumor, seen rather by the neurologist than by the surgeon, in which I take it the importance of this syndrome lies. We have not as yet found any cases of this particular syndrome either complete or incomplete, from brain conditions. On the other hand, we have one case, and doubtless will have more, as is the experience of Elsberg, Nonne and others, of cord compression from tumor with absolutely negative findings. What we recommend is to use this syndrome in connection with, and only in connection with, clinical findings, laying stress chiefly on positive findings. Dr. Skoog mentioned pressure, and I should have said more about the pressure. We measured the pressure in practically every case, and it was almost invariably normal. In one case the pressure was normal in spite of the fact that the patient had, besides his cord tumor which constricted the dorsal region, a brain tumor, which should have given a high pressure reading 400 mm. or more. This patient had 170 mm. pressure, which with our technic has been found to be normal. Dr. Ely's cases are exactly what would be expected. Syringomyelia certainly acts like tumor of an intramedullary nature. Dr. Mix's analysis was one of the best I have seen. But he does not seem to go any farther than did Mestrezat three years earlier; that is, he says the complete syndrome is necessary to be of clinical significance. But if we wait until then, I think in some cases we will have lost our chance; we have to use the incomplete syndrome in helping us to make an early diagnosis of compression. In one case where we had a series of punctures, the first one showed the syndrome in incomplete form. Operation was considered, but thought premature. A year and a half later the patient showed increase of symptoms, with increased intensity of the fluid syndrome; operation was then performed, and a tumor removed. It would have been done earlier had the significance of the incomplete syndrome been appreciated.

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CONSTITUTIONAL INFERIORITY

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In presenting the conception of constitutional inferiority it is necessary to emphasize the fact that the human mind is complex and intricate in structure, as well as from the point of view of organic and social heredity and its adaptation to the environment. It is important to bear in mind that in early life mental adjustment is purely instinctive and emotional. Furthermore, the volitional and intellectual faculties with the moral sense make their appearance rather late in mental development. While it is true that the whole mental organism is of a sensitive nature, nevertheless it evinces considerable plasticity necessary for adaptation. It is also to be remembered that because of these sensitive and plastic qualities, this organism is capable of receiving environmental impressions of favorable or pernicious character.

Of great significance is the fact that there is an underlying organic basis for the harmonious union of psychic forces necessary for the execution of a coordinate mental act, that is a healthy equilibrium of emotion, volition and intellection, which are controlled by organic heredity, and only in part modified by social evolution. It is not difficult to conceive how a congenital defect[1] in the affective, or volitional, or intellectual sphere or in all spheres may produce psychic instability, and indeed to such an extent as to cause a

1. Karpas: Psychic Inferiority, New York Med. Jour., 1913, xcvii, 594.

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maladjustment of varying degree and intensity. The fact that an isolated enfeeblement of the intellectual faculty only is not at all requisite to characterize constitutional inferiority per se is quite apparent. It is interesting to note that a lesion of one faculty may indirectly disturb the function of the entire mental organism.

In classifying constitutional inferiority, it is advisable to be guided by the individual fundamental disorder involved in each of the three faculties. Upon this basis we may divide mental inferiority into three large groups, intellectual, emotional, and volitional.

The intellectual type embraces a wide range of inferiority, which may differ in character and quality. Idiocy, imbecility, and moronism are examples of one extreme. Again, there are forms of relative intellectual deficiency which in a large number of instances are not of social evil, because volitional and emotional faculties are not so impaired as to bring about antisocial upsets.

The emotional form of mental inferiority presents well-defined characteristics. In some instances there is a deficiency or excess of emotional activity of such a nature as to produce distinct types of personalities. The *mental calms* are a well-known class. They are seclusive and secretive, rather timid and self-absorbed, indulge in dreams and the real world is of very little source of interest to them. Some of them are oversensitive, suspicious, and chronic masturbators. Such a type of personality was termed by Hoch as "shut-in" and is found in a large number of cases of dementia praecox. However, some of them never develop a psychosis or a neurosis, but they exhibit some difficulty to adjust themselves to their environment.

On the other hand, there is another type of personality which is of a highly vivacious nature, and of vacillating mood. Such persons are loquacious, rather keen, and manifest lack of capability to suppress or repress their feelings. This form of mental make-up

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is met in manic-depressive insanity, which was also described by Hoch.

In addition to these two striking types there is another one in which emotional instability is the fundamental disorder. To this class belong the nervous and hysterical temperament and a hypochondriacal and neurasthenoid disposition. To be sure, such individuals evince considerable instability and some of them develop definite neuroses or psychoses. Particularly it is true of the nervous temperament, which in addition to syphilis or alcohol develop psychoses peculiar to these etiologic factors.

Another important type of an abnormal personality which is determined by perverted feeling is known as the psychopath, or, in popular language, the crank. I prefer to call it a congenital psychoneurosis, because it has so many earmarks of an actual neurosis, however not amenable to radical treatment. Such persons present definite psychopathic characteristics which express themselves in oddity of conduct, vagaries in dress, asocial tendencies and fantastic ideas. As a class they are unstable and are not wage earners. They appear intellectual, but their education is only smattering and superficial. They are failures in life, the Utopian fancies in which they indulge never or rarely take a constructive or practical course. As a rule they do not commit gross crimes, except some sexual offenses, which they justify on the ground of the moral code peculiar to their kind. They make themselves conspicuous, but they are tolerated in society to some extent because they seldom come in collision with the law except in extreme instances in which their sociologic beliefs are at variance with the existing statutes.

Of all the other forms of constitutional inferiority none is as significant as the volitional type, because of its great social import. The underlying disorder is lack of development of will-power, feeling and moral sense. And to quote Maudsley.[2]

2. Maudsley: Responsibility in Mental Diseases, p. 184.

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He has no capacity of true moral feeling; all his impulses and desires, to which he yields without check, are egoistic; his conduct appears to be governed by immoral motives, which are cherished and obeyed without any evident desire to resist. There is an amazing moral insensibility.

There are no apparent intellectual disturbances according to the usual gross tests. However, on more searching analysis, one finds considerable superficiality, apperceptive faculty is weak, the enfeebled judgment is quite manifest when confronted with difficult situations or tasks and the stock of knowledge takes an egoistic trend. In the words of Maudsley.[2]

The intelligence is often acute enough, being not affected otherwise than in being tainted by the morbid feelings under the influence of which the persons think and act; indeed they often display an extraordinary ingenuity in explaining, excusing, or justifying their behavior, exaggerating this, ignoring that, and so coloring the whole as to make themselves appear the victims of misrepresentation and persecution. Their mental resources seem to be greater sometimes than when they were well, and they reason most acutely, apparently because all their intellectual faculties are applied to justification and gratification of their selfish desires. One cannot truly say, however, that the intellect is quite clear and sound in any of these cases, while in some it is manifestly weak.

In formation of habit and character, volition, emotion, and the moral sense play a very important part, and this explains why in these individuals the habits are faulty and the character is altered. They are deceitful, given to lying, contrive numerous schemes to cheat and have no control of their primary impulses, such as the sexual and nutritional instinct. A marked autoerotic feeling is in the foreground, which may assume different phases and forms. The altruistic tendency is very superficial, or not developed at all. In the attempt to adjust themselves they follow the path of least resistance, and because devoid of moral sense and deficient in will-power and feeling, they commit all kinds of antisocial deeds, which are frequently determined by temptation, suggestion and environment. However, the last invariably shows a certain degree of inconsistency between the act and the actual stimulus.

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Were we in possession of concrete tests to gage such an abnormal mental state, we would have very little difficulty to demonstrate that the emotions and volition are of a distinctively primitive type; in other words, we are dealing with a psychic malformation in which only one part of the mental organism reached a degree of maturity.

The clinical varieties of the volitional form of constitutional inferiority are many and indeed in some instances they remain undetected till they come in conflict with the law. It is significant to note that the more atrocious the crime, the more difficult to convince the layman or even the physician that such a crime resulted from an abnormal mind.

It cannot be too strongly emphasized that antisocial tendencies which are recurrent in nature and which are not adequately modified by environmental influences are invariably determined by a psychopathic condition. In this connection the question of mental responsibility may be introduced. Do such persons have a full knowledge of the nature and quality of the crime they have committed? If an intact mentality is determined by one faculty only, intelligence, and the other two, emotion and will-power, are to be disregarded, then such a sophistic conception of mental responsibility may appear plausible. However, according to modern psychology an intact mentality must depend on the harmonious balance of the three faculties, emotion, volition and intelligence; then how can a moral defective be truly responsible for a crime, when in reality he is constitutionally incapable of differentiating between right and wrong? The sooner we get away from the old conception of mental responsibility, the sooner we will come to a better understanding of the underlying factors controlling such abnormal mental state. The persistent inability on the part of the individual to conform with the moral law is sufficient proof that the inherent defect lies not in the environment but in the mental constitution.

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In a broad sense, the volitional form of constitutional inferiority should be regarded as a psychosis, because there is a marked element of maladjustment present. It is a malignant form of mental aberration and does not yield readily to rectification.

This is not a new disease, but it was recognized by the English physician Prichard[3] as early as 1835, when psychiatric progress was at the low ebb, anatomic knowledge of the nervous system was deficient, and psychologic investigations were permeated by the atmosphere of crude philosophic dogmatism. Prichard termed this peculiar mental affection moral insanity. Later investigators, while differing in the nosologic conception, yet admit the occurrence of such an abnormal mental state. Some preferred to term it moral imbecility, moral idiocy, and constitutional inferiority. It is of little practical interest what such a pathologic mental condition may be designated, the fact remains that this malignant form of mental aberration is of relatively frequent occurrence and is a great social evil. A large majority of the criminal class, tramps, vagrants, prostitutes, chronic alcoholics and other degenerates, belong to this group.

The following case is of interest:

A. is 12 years of age, born in England, and comes apparently from a healthy family. He was brought to the mental hygiene clinic because for the past eighteen months he has been continually stealing.

There are no neuroses, psychoses or mental degeneracy in the family or its collateral branches.

The patient is the second of five children. Labor was instrumental because of the child's large head, which was relatively out of proportion to the rest of his body. There is no history of convulsions or fainting attacks, but he suffered from enuresis for many years. He sustained several injuries to the head in early childhood. At the age of 7 he had membranous croup and at 7½ years of age suffered with chorea. He had measles, but no meningitis.

He entered kindergarten at 3 and later attended school; he failed only once in promotion, but this was ascribed to poor deportment.

3. Prichard: Treatise on Insanity, London, 1835, p. 12.

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The mother thinks that following the attack of croup he began to show changes in his mental condition, when he became restless, fidgety and stubborn. However, he is said to be good natured, but at times appears revengeful. Otherwise he is bright intellectually. He plays with boys and has many friends. He reads books and spends his evenings at home.

For the past eighteen months he has been given to stealing from his family and strangers. He takes everything he can turn into money. He gets up at night and steals money from his parents or from a stranger who happens to stay in the house. In addition, he lies a great deal and has absolutely no control over himself, although he realizes that it is wrong to take things which do not belong to him. Once he was arrested, but because he showed good intellectual endowment the mother was advised not to commit him to a reformatory.

Mentally the patient makes a fairly good impression. He appears bright and according to the intellectual tests is not feebleminded in the true sense of the word. When questioned about his acts he shows very little embarrassment. He admits that it is wrong to steal, but he could not help it, in his words: "I cannot resist the temptation, I want to get things." He knows that for stealing he will get a prison sentence. Despite the fact that he was reading very diligently of the recent trial and that he was told by his mother that he is just like the accused, nevertheless he makes no attempt to control himself. He states that with the money he steals he buys candy and spends it in other frivolous manner.

From the physical point of view, except a large head he shows no other abnormalities and there are no neurologic signs.

With this brief outline of the fundamental conceptions of constitutional inferiority, we may now briefly consider the question of the care and treatment of such patients. It is needless to state that the most important thing is to detect those forms of mental abnormalities in the early development stages, when simple adjustment may be undertaken. The normal mind is elastic and finds little difficulty to adapt itself to certain surroundings, whereas the abnormal requires an artificial environment in the process of adaptation. It is interesting to recall Huxley's[4] conception of education. In his own words: "What is meant by education is learning the rules of this mighty game. In other words, education is the instruction of the mind

4. Huxley: Liberal Education, p. 83.

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(intellect[5]) in the laws of nature, under which name I include not merely things and their forces, but men and their ways." He compares the world to a chessboard, the pieces to the phenomena of the universe, and the rules of the game he calls the laws of nature. The successful player is the one who knows the chessboard well, and has a good knowledge of the laws of nature. It may not be far fetched to say that the integrity of mental health depends on the knowledge of the laws of nature and ability to utilize them correctly in the adjustment of one's environment, the chessboard. If the chessboard is too complicated for one to play his game, why encourage him to make these mistakes repeatedly to his own detriment and to the detriment of society? In such cases give him a simple chessboard to play an uncomplicated game which does not overtax his mentality.

Briefly stated, prophylaxis in the plastic stage is the important measure, and special pedagogic methods, religious and moral influence, environmental reconstruction and physical and manual training are necessary in the process of readjustment and reeducation in such cases.

For the fully developed cases of the first and second group the actual care and treatment present less of a problem than the last class, inasmuch as many of them can be cared for in institutions for the insane or the feebleminded.

The volitional form of mental inferiority exerts directly and indirectly a pernicious influence on society. The fully developed cases with definite antisocial tendencies are incarcerated in protectories, prisons, reformatories, etc., but this in itself does not solve the problem in so far as such treatment is only symptomatic and not radical. Persistent and recurrent attacks of antisocial upsets should not be ascribed to sociologic

5. Intellect is used by Huxley; mind is substituted by the writer because it expresses the entire structure and function of the mental organism. In education the emotional and volitional spheres are just as important as the intellect.

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conditions, but deeper stratum for such abnormal reaction should be sought in one's mentality. It is manifestly important to analyze crime from three angles: First, we should obtain a complete knowledge of the psychologic background from which such a crime emanated, and ascertain the individual from the point of view of heredity and constitutional make-up. Second, we should determine the character and nature of the committed act and the frequency of its recurrence. Third, we should ascertain whether such a crime was determined by environmental motives. In many instances the antisocial deed gives the first clue of the person's defective mentality and indeed this is very important to establish in order to save society from such detrimental influences. Just the same as the homicidal paranoiac is a dangerous member of the community, so is the moral defective, and should be treated in a similar manner. They are both malevolent, and society should be protected against them. A deeper appreciation of this abnormal type of mentality will necessarily create ways and means of establishing institutions for such individuals and at the same time change our old conceptions of mental responsibility, and control further progress of vice, crime, and other forms of degeneracy.

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ABSTRACT OF DISCUSSION

DR. FRANK P. NORBURY, Springfield, Ill.: I would like to add a few remarks from the medicolegal viewpoint, when we are confronted with this type of constitutional inferiors in the so-called malingerers. As Dr. Karpas has said, these are the individuals who, when confronted with a difficult situation, seek the easiest way out and that within their own limitation. Malingering simply is a way out oftentimes. This not only occurs in civil cases, but I have been concerned with three cases of murder wherein malingering was attempted to be carred out as a possible defense for connection with the crime. The insanity, however, being assumed after commission of the crime. We proved the understanding of responsibility on the part of the defendant as outlined by criminal law. When that legal test was applied these

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individuals were convicted as murderers and a life sentence was imposed in two cases. The third case is still pending, and the fourth case is one that reached our supreme court on some technical errors in the trial. The man was sentenced for life but on account of the errors the case came to the Supreme Court of Illinois for review and retrial based on a technicality involving this very point of malingering. However, I do not know whether the case will ever come to trial, because when brought back the defendant made his escape and so far has not been apprehended. But he is constitutionally inferior in the emotional, the volitional, and the intellectual aspects of life. In one case that I had under observation, a man being tried for forgery in the United States Court assumed insanity as his defense. He truly was a malingerer. We all know that the malingerer as we see him, no matter whether involved in a criminal procedure or some milder offense, or even when attempting to deceive in some other way, is of an inferior type. This to him is the way out of a difficult situation and offers also an opportunity for him to make his impress on others to help him out of some situation. As for instance, begging alms or through interest in other affairs.

DR. M. J. KARPAS, New York: Most cases of constitutional inferiority of the volitional type do not malinger, as a rule. When they are under restraint they are perfectly willing to tell their whole life history and, indeed, this is rather characteristic in this group of cases. I must confess that I have not yet encountered a malignerer in this class of cases in the true sense of the word.

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EXPERIMENTAL STUDIES OF INJECTION OF THE GASSERIAN GANGLION

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AND
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A perusal of the contemporaneous literature leads to the conclusion that gasserian ganglion operations and intraneural or intraganglionic injections of alcohol are the procedures of selection for the treatment of trifacial neuralgia.

The technical difficulties of operations on the gasserian ganglion or its sensory root and the relatively high mortality attending such operations, except in the hands of a very few surgeons, made welcome a procedure which offered at least alleviation without danger, disagreeable sequelae or great difficulty.

The injection of pain deadening and nerve destroying substances into the trifacial nerve dates back to 1874, when Bartholow reported successes with the injection of chloroform. Successful results with the injection of osmic acid were recorded through a period of years following Billroth and Neuber in 1884, by Eulenberg, Shapiro, Bennet, Murphy, Wright and others.

It was not until Schlösser reported the results of his experiments in 1903 that persistent efforts were inspired for the development of a technic for successful alcoholization of the fifth nerve. About the same time further impetus was given by the work of Ostwald, Levy and Baudoin. The technic of the latter two was adopted by Patrick, Hecht, Sicard, Harris

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and others. The development of the technic and the reports of its successful employment by these workers has placed this procedure permanently among the methods employed for the effective relief of trifacial neuralgia.

It is not within the scope of this paper, which deals with a technical problem, to describe the details of the development of methods of injection and their results.

Suffice it to say that in the hands of operators who have had wide experience and who have developed consummate skill, all these methods are uncertain. As Patrick[1] says, "It required little experience or reflection to realize that no one can uniformly touch with a needle a given point 4 to 5 cm. (2 inches) below the surface."

The uncertainty of prevailing methods led us to pursue some experimental work on which this paper is based. We are concerned with reaching the gasserian ganglion through the foramen ovale. Part of the experiments deal with a route which Harris uses in his technic. He, with Patrick and others, employs with little modification the course adopted by Levy and Baudoin. The needle is inserted in the sigmoid notch from 1.5 to 2.5 cm. anterior to the anterior root of the zygoma or from a point immediately in front of the preglenoid tubercle, to one a centimeter farther forward. The needle is directed somewhat backward and slightly forward, and at 4 cm. should reach the nerve. The needle arrives at the foramen ovale after having passed through the skin, subcutaneous tissue, the zygomatic insertion of the masseter, posterior portion of the temporal tendon, superior border of the external pyterygoid muscle and lastly in the front of the temporo-maxillary articulation. Patrick[2] has called attention to the fact that:

1. Patrick, Hugh T.: The Treatment of Trifacial Neuralgia by Means of Deep Injections of Alcohol, THE JOURNAL A. M. A., Nov. 9, 1907, p. 1567.
2. Patrick, Hugh T.: The Technic and Results of Deep Injections of Alcohol for Trifacial Neuralgia, THE JOURNAL A. M. A., Jan. 20, 1912, p. 155.

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A really valuable landmark is the external pterygoid plate. The foramen ovale, at least in the vast majority of cases, is directly back of this and at about the same depth. Hence I aim to strike this plate close to its posterior edge and then work backward until the needle slips by it. Then I have every reason to believe that the point of the needle is just in front of the nerve and at about the proper depth.

Patrick gives a very valuable guide to be used in determining the approximate depth of the foramen from the surface. Analysis of his measurements in eighty cases indicates the following: When the interzygomatic diameter is 5 inches, the nerve will be found at 4 to 4.25 cm. When the interzygomatic is 5½ inches, the nerve is apt to be at over 4.5 cm.

If one follows these various details of procedure, the needle may be inserted to a place in very close proximity to the foramen, and we are concerned with making this proximity as great as possible.

A study of the anatomy of the parts about the foramen ovale shows that along the line of the sphenopetrosal fissure the edges of the adjacent sphenoid and petrous-temporal bones are beveled "so as to form a groove which extends from the root of the inner pterygoid internally, to the inner side of the base of the alar spine externally . . . in the groove is lodged the cartilaginous part of the eustachian tube. . . . Between the root of the external pterygoid plate and the alar spine there are two foramina which lie directly in front of the sulcus of the eustachian tube. Of these the larger and anterior is the foramen ovale." (Cunningham.)

A view of a sagittal section of the skull at the plane of the foramen ovale shows that it is directly in front of, above and internal to the eustachian tube. A study of both sides of fourteen skulls showed that however the groove for the eustachian tube varied in its course, the bone separating the sulcus from the foramen varied in thickness but from 1 to 3 mm.

It follows that to successfully introduce a needle into the foramen ovale it is necessary only to visualize

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the eustachian tube and to establish certain angles and lines which would by means of the fluoroscope enable one to determine when the point of the needle is in front, above and external to the eustachian tube at a proper depth.

The visualization of the direction and approximate location of the eustachian tube is easily accomplished by the introduction of a eustachian catheter with the assistance of a naso-pharyngoscope (so that one is certain of having entered the ostium), and by inserting through the catheter a bougie made of fuse lead or one made of flexible spirally wound gold wire. This, when it is inserted to a depth of about 1 inch beyond the end of the catheter, is about at the beginning of the isthmus of the tube at its entrance into the osseous canal. The course and position of the tube can now readily seen by means of the fluoroscope (Fig. 2).

The rays should enter the head at the opposite occipitoparietal protuberance at an angle of 120 degrees to a coronal section, and at an angle of 45 degrees to a sagittal section of the head. Under these conditions, a needle introduced at the sigmoid notch and taking a direction approximately at an angle of 20 degrees to the sagittal suture and 130 degrees to the coronal section would be directed toward the foramen ovale and be projected on the screen as traveling downward toward the eustachian tube, at an angle of 90 degrees from the point of entrance.

If the needle is introduced in this direction to a depth determined by computation from the intertubercular diameter, and does not cross the shadow thrown by the eustachian bougie, it is at its proper depth.

If the rays now be shifted so that they enter in the direction of the needle, the butt of the needle should appear above the eustachian tube, determining its proper height. This leaves undetermined the anteroposterior location of the foramen. The bony structure seen through the inferior border of the zygomatic surface of the great wing of the sphenoid throws a

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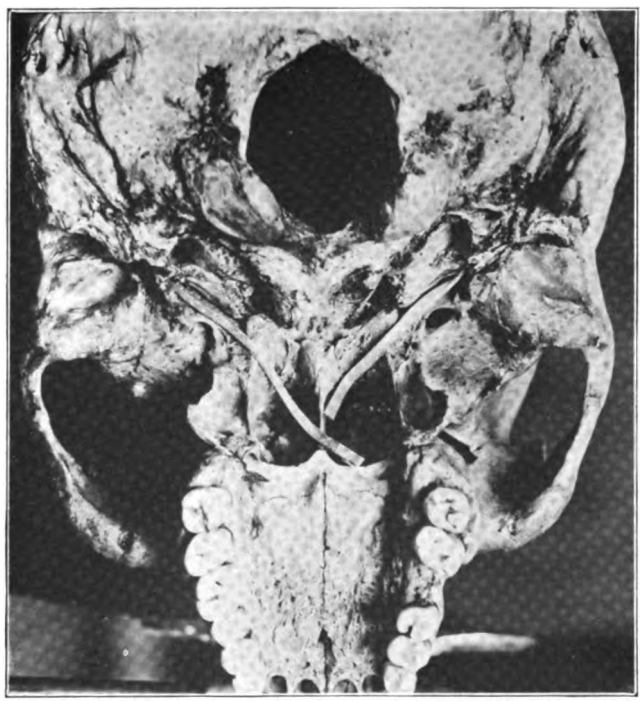

Fig. 1.—Lead strips in the groove of the eustachian tube, showing the foramen ovale immediately external and anterior to it.

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Fig. 2.—Needle in foramen ovale and eustachian bougie in position. The needle can be seen entering the foramen on the linear shadow cast by the structure through the inferior border of the great wing of the sphenoid.

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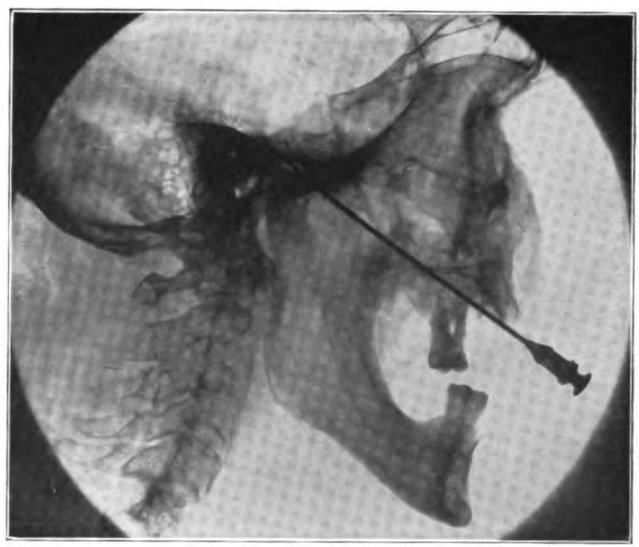

Fig. 3.—Needle inserted through the foramen, resting on the anterior border of the petrous portion of the temporal bone.

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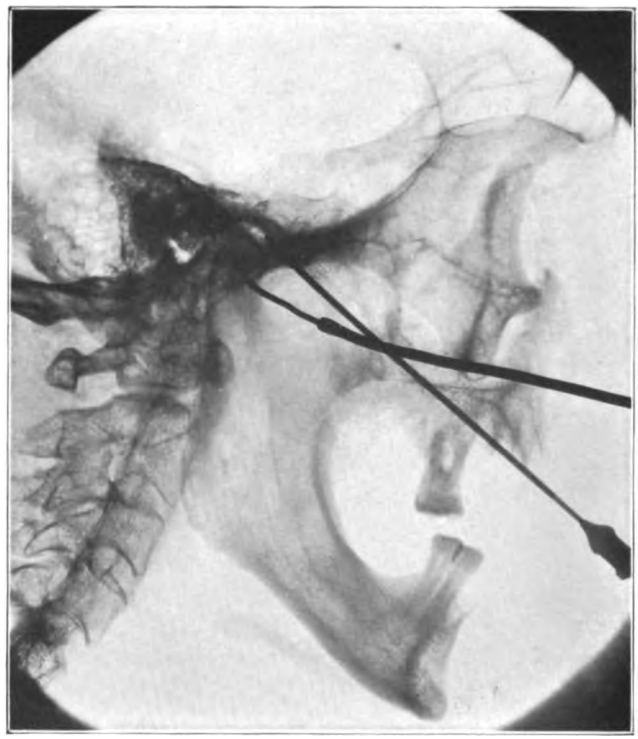

Fig. 4.—Needle in the foramen ovale, the eustachian bougie in place to indicate the proper height.

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shadow running anteroposteriorly and upward and when visible, the foramen ovale may be found somewhere along its course.

An approximate formula may be formed. If under the above conditions a needle be introduced to a point above the eustachian tube, and to a depth not crossing it, it can be caused to enter the foramen ovale by directing it at points along the described line from before backward. When this line is not visible, the needle may be directed from 0.5 to 1 cm. above the shadow of the eustachian bougie.

A large number of factors entering into the optical physics of the formation of the shadow of the eustachian bougie make the distance between the shadows of the eustachian bougie and foramen ovale vary to such a degree that the actual distance cannot be computed.

On cadaver heads this method offers such assistance that a needle can be successfully introduced into the ganglion on each attempt. When, however, we are dealing with live subjects, a new factor is introduced which for the time precludes the possibility of the employment of this method. We attempted this method in three cases and found that the secondary rays developed in the tissues, principally the brain, diffused the shadow of the slender needle to such an extent that proper methods of diaphragming must first be developed that will eliminate these rays before the fluoroscopic projection of the eustachian tube may be employed as a means of accurately locating the foramen ovale.

Failing in this direction, we began to study another route.

Härtel[3] has given us a route which has made the approach through the foramen ovale more practical and useful. To Grinker,[4] who first made Härtel's method known to American readers, we are indebted

3. Härtel, F.: Arch. f. klin. Chir., c, No. 1.
4. Grinker, Julius: A New Method of Treating Neuralgia of the Trigeminus by the Injection of Alcohol into the Gasserian Ganglion, THE JOURNAL A. M. A., May 3, 1913, p. 1354.

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for some of the following description. The needle is introduced through the cheek opposite the alveolar process of the second upper molar tooth. It is guided by means of a finger in the mouth so as to pass between the anterior ramus of the lower jaw and tuberosity of the superior maxilla. "Causing the needle to sweep around the buccinator muscle, the operator pushes it onward between this muscle and the masseter, coronoid process of the lower maxilla and the temporal muscle externally, into the zygomatic fossa. Continuing in the direction upward, backward and slightly inward, the needle now reaches the broad under surface of the great wing of the sphenoid, having pierced the external pterygoid muscle, which fills the zygomatic fossa."

According to Härtel, the needle points to the pupil of the eye of the same side and on lateral view the needle points to the articular eminence on the zygoma. The needle engages the foramen ovale at a depth of about 6 cm. from the point of entrance.

Maes[5] has found that the foramen was more accessible from a point behind the last molar tooth of the lower jaw than from the point given by Härtel. From a study of forty skulls we found that projecting a pin through the foramen ovale, hugging the anterior border of the petrous portion of the temporal bone, the point of entrance would fall in most instances at a point between the second and third molar of the upper jaw.

The depth to which the needle should be introduced may be more accurately estimated by using the data we have derived from an examination of both sides of forty skulls. The distance from the point of entrance of the needle to the preglenoid tubercle, or the alveolar tubercular measurement, was greater than the distance from the point of entrance to the foramen ovale or alveolar foraminal measurement in all but two instances. Of these one was an anomalous skull, and the other, one in which the intertubercular measure-

5. Maes, Urban: Surg., Gynec. and Obst., 1915, xxi, 449.

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ment exceeded 5½ inches. In all other skulls it was found that the alveolar foraminal was less than the alveolar tubercular measurement by from 0.1 to 0.9 cm. Eighty per cent. of the cases showed a difference of less than 0.6 cm., 50 per cent. less than 0.4 cm. Of the cases showing a difference of over 0.6, many occurred where the intertubercular diameter was less than 5 cm., and some where the alveolar tubercular measurement exceeded 5.5 cm. The depth of the foramen from the zygoma did not bear a constant relation to the alveolar foraminal measurement, except in that where the foramen was deeply situated the alveolar foraminal measurement was often inclined to be greater than usual.

The anterior border of the petrous portion of the temporal bone, when the head is viewed exactly laterally through the fluoroscope, throws a shadow easily seen in living subjects. This shadow forms a line which is a direct projection of the shadow cast by a needle entering a point between the second and third molar of the upper jaw. If, therefore, a needle be introducd here and pushed, according to Härtel's directions, to a depth computed by our measurements, it will be approximately at the foramen ovale. If now the needle be directed so that a projection of the shadow would follow the shadow of the anterior border of the petrous portion of the temporal bone, or 1 or 2 mm. below it, the anteroposterior and superior planes of the foramen ovale will be accurately located. The internal and external planes of the foramen ovale cannot be located by the fluoroscope, and one is guided by Härtel's direction to point at the pupil of the same eye and by the pterygoid bone. The superior inferior plane may be further determined, if necessary, by employing a shadow of an opaque eustachian bougie, when the needle should be above it, and here the secondary rays do not interfere with the visibility of the shadow of the needle.

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In our experiments, employing both sides of the fourteen heads, no difficulty was experienced in introducing the needle into the foramen ovale on each attempt. It might be said that the introduction of a needle into a given point of a cadaver which cannot bleed, suffer pain or experience injuries of various important structures, is quite a different matter from accomplishing the same result in the living subject.

We have had only one opportunity of using this method on a living subject. Here the lower and middle branches of the ganglion were easily reached and injected, with immediate relief of pain and good analgesia.

In conclusion we may say that employing the shadow cast by the anterior border of the petrous portion of the temporal bone, on a fluoroscope, as a line of orientation injection of the gasserian ganglion by Härtel's method is made more certain.

We wish to express our gratitude to Profs. J. Gordon Wilson and S. W. Ransom for placing at our disposal the anatomic material on which our experiments were performed.

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ABSTRACT OF DISCUSSION

DR. HUGH T. PATRICK, Chicago: It remains to be seen whether Dr. Pollock's method will prove to be a practical one to be used in the majority of these cases. The method of Härtel, under proper guidance, perhaps according to the method which Dr. Pollock has devised, may prove to be an entirely practical and practicable procedure; but I must say that under the rules which Härtel himself originated, I never had the courage to attempt to inject the Gasserian ganglion.

I may exaggerate the dangers a great deal but I do not really want to demonstrate to myself that it is a dangerous procedure. The operation has, of course, been done successfully a number of times, and I hope under the scientific guidance of Dr. Pollock we may be able to do this accurately and without danger. And this is a consummation devoutly to be wished. Injection of the ganglion by the Harris method is disappointing to me. I have tried it a number of times and occasionally the needle goes through the foramen ovale and reaches the Gasserian ganglion or its vicinity with a facility that can be compared with putting one's hand in his pocket. And the next time the needle absolutely does not find the foramen at all.

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DR. JULIUS GRINKER, Chicago: While in Berlin in 1902-03 I saw Härtel demonstrate his method, which was new then. I also saw the cases as he demonstrated them before the Berlin Surgical Society. There were five cases, each one having been afflicted with a very formidable sort of tic douloureux and each one having received a number of peripheral nerve injections without benefit. Relief came promptly after Härtel's injections. The first injections were made, not to relieve neuralgia, but to anesthetize the face for operation. For this purpose Härtel uses small doses of novocain introduced into the Gasserian ganglion, which permit operation on any part of the face without a general anesthetic. It was the ease with which he succeeded in anesthetizing the face that led him to adopt the injection of alcohol directly into the ganglion as a means of giving relief for trigeminus neuralgia. My own impression of the method is that it is not more difficult than injection by the Boudouin-Levy method with which Dr. Patrick made us acquainted. When first shown to me, I thought it a rather formidable procedure—to take a needle of such large caliber and bore a hole in the patient's face. Nevertheless we tried it and succeeded in giving relief to many unfortunate sufferers. Härtel uses a very fine needle, so fine indeed, that it matters little whether you introduce it twenty times or only once. It does no damage if you follow his directions. Nevertheless, I believe a simplified method would be a great advantage. Personally I do not so much fear the difficulty of getting into the ganglion, as the consequences of having entered the ganglion, namely, the corneal ulceration. As a successful gasserectomy done by the surgeon is in many instances followed by ulceration of the cornea, so do we get a similar result after successful injection of the ganglion. And it is this apprehension which has really prevented me from repeating the injection more than twice. In short, I fear the after-effects of a successful injection much more than the difficulty of getting into the foramen.

DR. M. A. BLISS, St. Louis: I have felt that the injection of alcohol was a more or less temporary measure. The cells of the ganglion are much more resistant to alcohol than are the fibers of the trunk, according to the observations I have seen quoted. I would like to ask whether in any successful injection of the Gasserian ganglion the anesthesia and the relief from pain is reasonably permanent.

DR. CHARLES R. BALL, St. Paul: For about eight years I have been making these injections by the Boudouin-Levy method which Dr. Patrick first brought to this country. Injection of the ganglion is no trivial procedure. The procedure is to be entered on with a proper sense of appreciation of the danger connected with it. In the first place we must have the right sort of a needle. I sent to Berlin and procured Härtel's injection outfit; since then I have been using the Härtel needle in all my injections. It has the disadvantage

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that after you start your direction, the needle is so fine that you cannot change it; therefore if you do not enter the foramen you have to withdraw the needle and start over again. Another objection to the fine needle is that a little bit of blood is liable to cause it to become occluded, particularly if you have squeezed a few drops of alcohol through the end of the needle. Alcohol coagulates blood very readily, and it is most distressing sometimes to find that you are in the correct position, but that the needle is occluded.

In getting into the gland I have had the experience related by Dr. Patrick. When I heard Dr. Harris describe his method it seemed good, so I practised on a cadaver and found no difficulty in entering the foramen ovale from the side according to the Harris method, also no difficulty in entering the foramen ovale from the front according to the Härtel method. But I noticed that many times when I went into the foramen of a living subject this did not mean that I had got into the ganglion by any means, but that the end of the needle was under some dense tissue, and this tissue prevented me from injecting the ganglion with alcohol.

The best ganglion injection I ever made was an accidental one. I was trying to inject the third branch of the nerve according to the Levy-Boudouin method. I introduced the needle to the point where I thought the exit of the third branch ought to be and injected about 1 cc. of a 90 per cent. solution of alcohol. The patient immediately went into collapse, became unconscious, had paralysis on that side of the face, and I thought she was going to die on the table. She was over 80 years old. The facial paralysis was the most complete I ever saw, but this did not bother her a bit; we had a beautiful anesthesia of the three branches of the fifth nerve and she was highly pleased and delighted with the results. Last winter I had one of the most intractable cases that I have ever encountered. I am sure that I entered his foramen ovale according to the Härtel method on two different occasions and injected alcohol. On one occasion I got facial paralysis and nystagmus, anesthesia of all the branches of the fifth, and temporary anesthesia of the cornea, and yet, with the exception of the partial anesthesia of the cornea, the pain still continued.

Dr. Ernest Sachs, St. Louis: Dr. Ball is frank enough to tell us of the untoward accidents. I should certainly hesitate to undertake any procedure when I had such complications. I judge that what happened in Dr. Ball's case was that when the alcohol was put into the cavum meckeli it went where the cerebrospinal fluid goes; when you open the cavum meckeli you always get discharge of cerebrospinal fluid. I have had no experience with injection of the ganglion; I would not think of doing it or trying it. Gasserectomy, when carefully done, is not the dangerous operation that it is generally supposed to be. About a month ago

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Dr. Harvey Cushing reported about 150 gasserectomies without a single death, no complications, except one or two corneal ulcers which healed, and no blindness. It would seem to me that results of that sort are far better than doing an operation which is absolutely in the dark and very dangerous.

Dr. George Morris Dorrance, Philadelphia: In cases of operation on the face, we inject the ganglion with novocain to obtain the required anesthesia. We injected 300 cadavers, making 600 injections. In some cases we went under the ganglion and that made us fail; in others, over the ganglion, sometimes hitting only a part of the ganglion. I usually get it the first or second time and it comes easy after doing a few. You should, of course, inject novocain before the alcohol. If you get complications, you do not inject the alcohol but withdraw the needle and make a second puncture. If the neuralgia is confined to the third division, you do not necessarily inject the ganglion, but the third division only. However, it is not difficult to inject the ganglion. We do have recurrences, but when the operation is done right along, the procedure is not so difficult as has been intimated by Dr. Sachs. Sometimes we do find a difficult case; sometimes the material will not diffuse through the ganglion easily. Be sure to use the novocain first. It saves getting into these terrible complications.

Dr. Lewis John Pollock, Chicago: It was not my intention either to provoke or to enter into a general discussion as to the merits of alcoholization of the component portions of the nerve nor the ganglion itself. I had felt, contrary to the impression of Dr. Dorrance, that injection of the ganglion was difficult, irrespective of any procedure known at the present time; and that if a method could be devised which would make it more certain and less liable to be followed by any untoward results, we would be doing some service. The dangerous zone in doing the Härtel injection lies not anteriorly, but posteriorly, inferiorly and internally. If the needle is kept so that it enters not more than one or two millimeters below the shadow of the anterior border of the petrous portion of the temporal bone, it cannot go either too far back or too low down. Therefore, it will strike either the external plate of the pterygoid or the large wing of the sphenoid and you can do no damage to the structures here. As to Dr. Grinker's apprehension concerning the production of corneal ulcers, one should, of course, mention this complication under the discussion of advantages and indications and contraindications of injection of alcohol, and other methods of surgical treatment of trifacial neuralgia as well. In reply to Dr. Bliss, I would state that the ganglionic as well as the intraneural injections are only temporary. But in experimental work done by Byrnes it has been found that the sensory cells of the ganglion itself do degenerate and that this

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practically puts the nerve out of commission, more so than injections into the nerve itself. Of course, it is not necessary to inject the ganglion by this method; you do not have to push the needle in all the way; you can inject the lower branch alone, and that is the branch which is difficult to inject. The middle branch is easily injected. And it is not necessary to include all the ganglion in the injection if one proceeds carefully; if one is guided by proper measurements he will be able to separate the injection of the third branch from that of the first. I was very glad to hear Dr. Dorrance bring out the point that one can inject the third branch alone.

Referring to Dr. Sach's remarks, from a review of the literature as well as from personal observations, I feel certain that these untoward accidents do not result in permanent damage and, further, that they occur but very seldom, and exceedingly seldom as compared with the general results obtained by all general surgeons collectively in operations on Gasserian ganglion or its sensory root.

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NEW METHOD OF INJECTING FACIAL NERVE FOR RELIEF OF FACIAL SPASM

GEORGE M. DORRANCE, M.D.
PHILADELPHIA

Because of the rather confusing definitions found in text-books, particularly those of a few years ago, I shall give a brief differential diagnosis between facial spasm and tic or so-called habit spasm before describing the treatment.

A second purpose in doing this is that the treatment of those two affections is entirely different, and while alcoholic injections are ideal in cases of facial spasm, they are entirely contraindicated in tics.

True facial spasm is a reflex act occurring in a single muscle or a group of muscles, and is thought to be due to some form of irritation in the muscles or the nerves of the spinal or bulbar arc.

Tics, on the other hand, are voluntary contractions which have become a habit. It is a psychoneurosis and it is not a spasm at all. Briassard believes that the synergic and coordinated musclar contractions of tic imply a cortical origin.

In facial spasm, the spasm is confined to the mechanism of the motor innervation of the face, and is devoid of voluntary or involuntary control; whereas in tics, the movements are always more or less under the control of the will, and subject to involuntary control under violent excitement or emotion.

The spasm of the true facial type is never a natural movement, and cannot be imitated either by the patient or by a second person. In tics, the movement, however exaggerated, is a natural one and can be imitated.

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As Patrick once said, "In spasm the movement is an anatomical disorder, in tics, a physiologic one."

At the onset of facial spasm, the spasm may be confined to only a part of a muscle. Later it takes the entire distribution of the facial nerve and no more, and is practically always unilateral.

Tics never involve only a part of a muscle, and rarely involve all the facial muscles at any given time, but take associated groups of muscles, varying these from time to time. It is often bilateral.

Facial spasms are painful, and incapacitate their victims, whereas tics give a certain pleasure or sense of relief, and never interfere with any of the bodily functions, as talking.

Spasms are found in the mentally strong, tics in the neuropathic type of persons in whom other mental stigmata exist.

There is another form of tic occurring after facial palsy in which the tic is synchronous with the winking. In this form, likewise, injections should never be made.

ALCOHOL INJECTION

My reasons for devising a new method for injecting the facial nerve are two:

1. The older methods are inaccurate (often requiring as many as ten punctures to hit the nerve).

2. The methods are dangerous, that is, the jugular vein is very apt to be hit.

I formed these conclusions after careful clinical observations and performing 300 injections of the facial nerve at the stylomastoid foramen on cadavers.

Through the courtesy of Dr. Greeman, I made a series of measurements at the Wister Institute. On fifty skulls, the distance between the stylomastoid foramen and that of the jugular varied from 0.2 to 1.2 cm., the average distance being 0.6 cm.

One can readily see how in the older method of simply holding the needle in a horizontal position and

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inserting the point into the stylomastoid foramen, that less than 0.5 cm. separates the point of the needle from the jugular vein.

Because of these difficulties and dangers, I devised a method whereby it would be impossible to hit the jugular vein, and yet permit one accurately to inject the nerve at its foramen of exit from the skull.

THE METHOD

The needle (trochar and stylet style) is 10 cm. long and 0.4 cm. thick, graded in centimeters. It is inserted at the angle of the jaw (the skin having previously been painted with 5 per cent. tincture of iodin and anesthetized with 0.5 per cent. novocain), and directed backward and upward until the point impinges on the base of the mastoid. The operator can make certain of this by feeling the point under the skin by the index finger, which is held over the mastoid.

The handle of the needle is elevated, and the point is depressed, the operator feeling carefully the way down, at the same time pushing the point backward and inward until the needle feels its way into the stylomastoid foramen.

The stylet is now removed. If any bloods flows out, no injection should be made, as the point may have pierced a small vein. The needle is withdrawn, cleaned, and another puncture made. If no bleeding occurs a few drops of alcohol are injected. If the nerve is hit succesfully, immediate facial paralysis occurs. About 2 c.c. more of alcohol are now injected.

The needle is removed. No dressing is needed.

As a rule, the patient complains of some pain, which is relieved by hot compresses.

The eye should be protected by goggles and washed out with boric acid solution at least twice a day.

For some little time after the injection, goggles should be worn when the patient is outside the house.

This differs from the fifth nerve injection in that we have no trophic changes or corneal ulcers. We must,

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however, remember that because of the paralysis, winking does not occur; hence the cornea is apt to become dry, owing to lack of proper irrigation from tears.

Moving the eye from the side and gentle massage will help to prevent any deleterious results. The face should be massaged daily to prevent atrophy of the muscles.

The method of injection appears to me to be extremely simple, and one or two hours' practice in the dissecting room to train the fingers in the sense of touch, a feature so essential in all injection methods, will render any physician proficient.

I have found from my records that the average depth of the needle was 5 cm.; but the distance varies according to the point of insertion and the amount of adipose tissue present on the face of the person injected.

By this technic, the entire distribution of the seventh nerve is paralyzed.

I have on several occasions injected one of the branches of the seventh nerve going to one or more muscles by injecting the branches anterior to the parotid gland.

It is much more difficult to inject the branches as the plane of the nerve must be hit, as alcohol does not diffuse through facial planes.

The results from the injection of the branches do not compare as favorably as those obtained by injection at the stylomastoid foramen. It appears as if it were necessary to rest the entire distribution of the seventh nerve to effect a perfect result.

RESULTS AND CONCLUSIONS

1. Most patients will willingly exchange a spasm for a paralysis.

2. The paralysis is only temporary. Voluntary motion slowly returns until complete function is restored.

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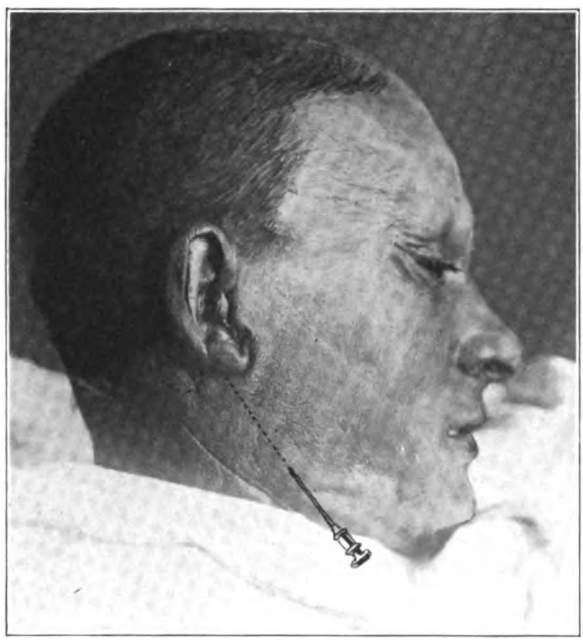

Fig. 1.—Showing needle in proper position.

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Fig. 2.—Showing needle in position on skull.

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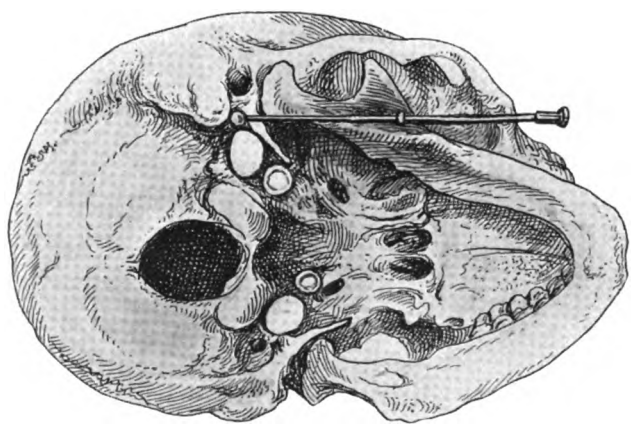

Fig. 3.—Note how the styloid process prevents the needle from going too far inward, thus preventing any possibility of hitting the jugular vein.

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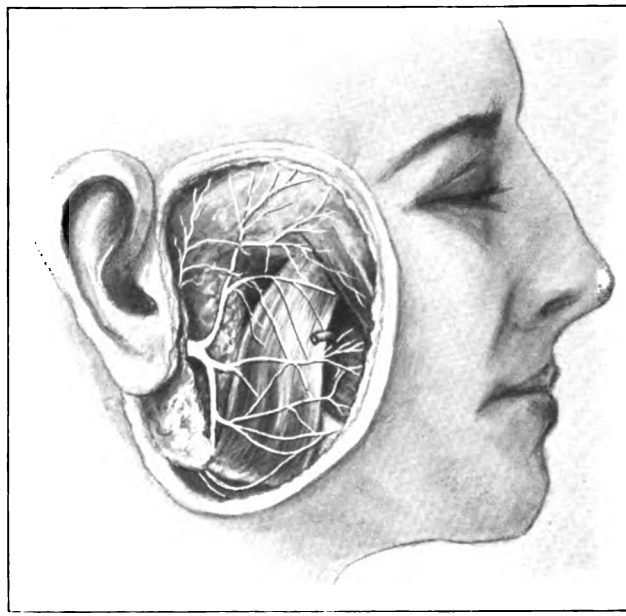

Fig. 4.—Distribution of facial nerve. Note the difficulties offered to the injection of the branches.

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3. The spasm never recurs at the time of the return of voluntary power, but varies from a month to several years. In some cases, it never recurs.

4. In the event of spasm recurring, patients are willing to have the injection repeated. (Return of power is slower with each subsequent injection.)

5. For injection at the stylomastoid foramen I use 2 c.c. of 70 per cent. alcohol; for injection of the branches, not over 50 per cent. alcohol, because of the danger of sloughing, which is prone to occur from the superficial injections in the loose fatty areolar tissue.

2025 Walnut Street.

ABSTRACT OF DISCUSSION

DR. HUGH T. PATRICK, Chicago: I am not in absolute accord with Dr. Dorrance's statement that tic never should be injected, although I have expressed the same opinion. But in saying this I do not mean at all that the differential diagnosis between tic and spasm is not perfectly distinct. They are not related affections at all except geographically. But I have been guilty of injecting one case of bilateral tic which was not in any sense a spasm. The patient was a commercial traveler, and he said this tic was ruining his business. He had an unusual, almost constant bilateral grimace which affected his eyes and the lower part of his face as well. I explained to him what this injection would do—that it would paralyze both sides of his face, and added: "There is absolutely no use injecting one side because then you would have paralysis of one side of the face and tic on the other side, when you would be more of a spectacle than ever." And he said, "For heaven's sake, do it!" I injected both sides, and, peculiarly enough, I struck both nerves on the first attempt. The result was startling. His face was absolutely devoid of expression, he could only partially close the eyes and he could not articulate any of the labials well. But strange though it may seem, the man preferred that condition to his tic. Fortunately the one injection was not very successful and he began to have some movement on one side in a day or two. Dr. Dorrance made one careless statement—that facial spasm is painful. It is not; it is tiresome, it is wearing, it is distressing, but not painful. I have often failed to reach the facial nerve, although I have in a measure approximated Dr. Dorrance's method by not any more going in at right angles to the face, but upward and backward. Even then I frequently have missed it. I entirely agree that the patients always prefer the paralysis to the spasm.

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DR. A. L. SKOOG, Kansas City: I agree with Dr. Patrick that these facial spasms are not painful, but they are disagreeable to the patient. I wish to ask Dr. Dorrance if he has had any bilateral cases which he has injected by his method. I have under observation now one case of bilateral spasm which I injected the first time about a year ago, not inject the facial nerve, the peripheral branches. The method I employed was to inject a number of times until I had secured what I thought was sufficient paralysis. On the first injection I obtained paralysis of one of the superior branches of the facial nerve, a complete paralysis which endured for a long time. The second series of injections was given about two or three months ago and again I obtained paralysis of the same branch. The result of peripheral injections in this case has been quite satisfactory, except that it had to be repeated in about eight or nine months.

DR. JULIUS GRINKER, Chicago: I should like to ask Dr. Dorrance to what depth he inserts his needle.

DR. GEORGE M. DORRANCE, Philadelphia: I insert the needle about 5 cm., the exact distance depending somewhat on whether people are thin or fat. Some of my patients complained of this condition being painful, but perhaps they did not know how to explain it. The condition kept them awake, and they described the sensation as one of pain. Perhaps it was merely their way of describing the condition. According to the literature bilateral facial spasm is very rare. I have never seen a case. The results of this treatment have been very satisfactory, for by this means we give the whole nerve rest. I would warn against the injection of too strong a solution of alcohol under the skin. In experimental work we have had a lot of sloughing of superficial fat and skin from using 90 per cent. alcohol and some dogs have died. If you inject a solution of about 50 per cent. strength you will not get sloughing of the superficial fat and skin.

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MYASTHENIA GRAVIS, WITH THYMOMA

W. A. JONES, M.D.
MINNEAPOLIS

The patient whose case history is here given presented himself at the City Hospital in Minneapolis in November, 1915. The first and cursory examination suggested the usual type of bulbar paralysis, but after the patient had been admitted to the permanent ward and studied more carefully, the diagnosis was changed to that of myasthenia gravis.

The family history was as follows: The mother was living and well at 89; the father died at 60, the cause of death not known; two brothers were living and well; one brother died of pneumonia; there were no sisters; the wife died at 38, cause of death not known; there were two children, both of whom were living and well.

The history of the antecedents, so far as the patient was able to remember, revealed no nervous or mental trouble in the family, on either the paternal or the maternal side. There was no history similar to the condition from which the patient suffered in any member of the family.

The personal history showed that the patient had always been a strong, healthy man, and had never been seriously ill prior to the onset of his present trouble. He had the usual exanthemata of childhood, from which he made good recoveries without sequelae. Twenty-seven years before he had an attack of malaria, but was able to be about, and was at no time seriously ill. He had no other history of illness. He had suffered no serious injuries of any kind, and never had been operated on. His habits had always been moderate. He used very little coffee, no tea, and no alcohol, but had been for some time a heavy user of tobacco, and gave a history of more or less persistent constipation. Prior to the attack his appetite had been good, and he had slept well. He denied any venereal trouble, nor could any be elicited from laboratory findings. He had an inguinal hernia, but it was not troublesome or annoying. He had occasional attacks of tonsillitis, and he had hemorrhoids.

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To the best of the patient's knowledge, he was in perfectly good health three years before. About May, 1912, following an exposure to cold while working in water as a ship's carpenter, he noticed within a few days a marked weakness of his neck muscles, and within two weeks he had developed a condition similar to the one from which he suffered when examined. The symptoms came on rather slowly, and were associated with slight pain and soreness in the muscles of the shoulder and the back of the neck, followed by a general muscular weakness, and particularly by an inability to close his mouth, to swallow solid foods, or to drink liquids. This condition progressed until the patient became very emaciated, and was almost unable to take nourishment at all. This illness lasted a few weeks, when he began to improve and recovered his strength; and within six months after the onset of his primary symptoms, he claimed that he was perfectly well, and during the interim he did not recall that he had felt any weakness of muscles or any inability to swallow or to speak.

Up to February, 1915, he was in his usual good health, and was able to attend to his work. During the month of February, 1915, without apparent cause, unless it was a slight cold, as he expressed it, following exposure, the above-named symptoms began to return. There was pain in the jaw, but not of a severe type. Gradually the muscles of the neck and throat became weak, and he was unable to move his jaw or to swallow or drink with his usual ease. At the time of his admission into the hospital in November, 1915, he had a slight ptosis of the right eyelid. The weakness that he complained of was most marked in his neck muscles, but the entire musculature was more or less involved, and during the time that he was under observation he was unable to walk, to make any marked exertion, or to eat a full meal. After a good night's rest the patient was very much better, but as the day went on his weakness became more marked. At the beginning of a meal swallowing and other movements of the jaw were comparatively free; but after a few moments the muscles tired, and he was unable to take further nourishment. It was difficult for him to blow his nose, and he was obliged to have his mouth closed and his chin held in position until he could force air through the nostrils. After talking for a few moments he was obliged to hold his lower jaw with the aid of his hand. Aside from this general weakness, and particularly of the muscles of the neck and jaw, and his inability to finish his meal, the patient felt fairly comfortable.

Neurologic examination seemed to show the mental condition to be normal for a man of his age. His speech was rather indistinct and slow, rather characteristic of his muscular weakness.

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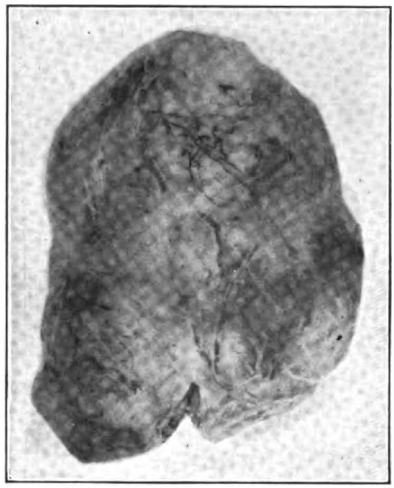

Fig. 1.—Photograph, natural size, of tumor removed at necropsy, situated in thymic region surrounded by firm capsule, with no perforations.

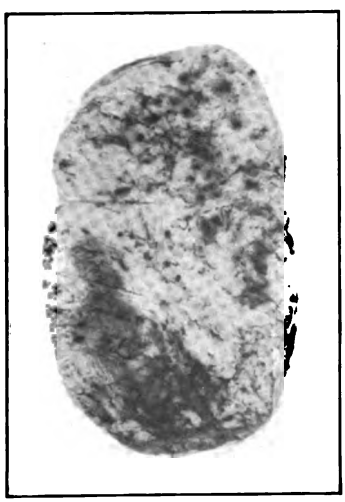

Fig. 2.—Cut surface of tumor shown in Figure 1. No lobulation; numerous hemorrhagic areas plainly visible.

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The first cranial nerve was normal. The second showed that vision was fair in both eyes and he had normal eye-grounds.

The ocular muscles, controlled by the third, fourth, and sixth nerves, were normal, with the exception that the right rectus was somewhat lax. He had no double vision and no nystagmus. The pupils, however, were irregular and slightly unequal, the right being larger than the left; both reacted sluggishly to light, but were normal for accommodation.

With the exception of a slight ptosis of the right eyelid, and the impairment of the external rectus muscle on the right side, there was nothing further to implicate the third and sixth nerves.

There was an apparent atrophy in both masseter muscles, and a marked inability to move the lower jaw. As a result of this involvement of the fifth nerve the mouth was open most of the time.

The seventh nerve presents no irregularities.

The eighth nerve showed that there was a moderate amount of deafness in the left side.

The tongue and palate were apparently normal. There was no facial or tongue tremor, except as was shown by his inability to swallow, and the fact that his speaking voice was below par, and any continued effort on his part to talk was accompanied by rapid muscular tire.

There was apparently no sensory disturbance to cotton, pinpoint, deep and superficial muscle sense, or vibration sense.

The biceps reflex was present and also the triceps. The supinator was active, and the patellar, active plus. The Achilles and abdominal and cremasteric were not obtained.

Coordination as far as was determinable was unimpaired.

The blood count was practically normal. A laboratory investigation of blood and spinal fluid showed the Wassermann negative. The urinary findings were also negative, eliminating any probable disease of the kidneys.

During the patient's entire stay at the hospital there was but little change from week to week in his condition. At times he felt better and stronger, and was able to move about in bed better at times; but there was no evidence of any marked change, with the exception of his dyspnea. On Dec. 10, 1915, he complained in the afternoon of a general fulness of his chest, an evident dyspnea. A hypodermic of $\frac{1}{200}$ grain of atropin was given, with the hope that it might relieve his distress. Ten minutes after the hypodermic was given he died suddenly.

A necropsy was made on Dec. 9, 1915, twenty-four hours after death, the report of which is as follows:

The body is well developed and fairly well nourished; pupils, 5 mm. in diameter and equal, no edema, no special marks.

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The peritoneal cavity contains no excess fluid and no adhesions. Intestinal surfaces are smooth and glistening. Appendix is 8 cm. in length and free. Diaphragm extends to the fifth rib on either side. Mesenteric lymph nodes are firm and slightly enlarged.

There are no excess fluid and no adhesions in the pleural cavities.

The pericardial cavity contains about 3 c.c. of thin, clear fluid.

The heart weighs 275 gm. The epicardium is smooth and clear. The mitral valve shows a slight diffuse thickening. There are no lesions in the other valves. The endocardium is smooth and clear. The muscle is pale and firm. The root of the aorta shows no lesions.

The left lung shows a firm, whitish thickening of the pleura at the apex; thickness, about 6 mm.; dimensions of area, 2 by 1 cm. The lungs crepitate throughout. They are very deeply colored with black pigment. Throughout the lungs is a considerable number of small miliary nodules of firm consistence. In the apexes the miliary nodules show firm fibrous tissue of a black color. The cut surfaces of lungs are fairly dry. The right lung weighs 375 gm., left 362 gm. There is an old healed apical tuberculosis of the lungs; old healed miliary tuberculosis of lungs.

The spleen weighs 130 gm. The capsule is wrinkled and slightly thickened. The pulp is fairly firm. The cut surface shows prominent lymph nodules.

The liver weighs 1,300 gm. The capsule is smooth and glistening. One small miliary nodule is noted on the anterior surface, resembling in structure those described in the lung. The cut surface shows no special markings. Lymphorrhagia are numerous in the liver, and a few were found in the adrenals.

The pancreas, gastro-intestinal tract and adrenals show no gross lesions.

The left kidney weighs 108 gm.; the right, 112 gm. They are of a dark red color. The capsules strip readily, exposing smooth surfaces. The cortices are about 7 mm. in thickness and of a dark red color.

The bladder and genitalia show no pathologic condition.

The aorta is fairly elastic. A few very small, elevated, yellowish areas are noted.

A tumor is found in the thymic region, just above the heart and immediately behind the sternum. It is embedded in loose connective tissue, and is not adherent to the sternum or to any of the thoracic viscera. Microscopic examination of the adipose tissue surrounding the tumor shows the usual senile thymic tissue. The tumor is easily shelled out of the connective-tissue envelope. It weighs 60 gm., is 6 cm. long, 3.5 cm. deep anteroposteriorly, and about 4 cm. in transverse direction. It is completely enclosed in a strong fibrous cap-

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sule about 0.5 mm. thick, which is not perforated at any point. The cut surface is of soft consistence, somewhat softer than the thymus of a child, and is studded with hemorrhagic areas. The cut surface is of a light gray color except in the portions containing blood. No lobules can be seen. (Compare Figures 1 and 2.)

Microscopically the tumor is seen to consist of fetal thymic tissue, that is, it shows epithelial cells in solid masses or arranged in a network among which there are varying numbers of small lymphocytes. The tissue corresponds to the fetal thymus at the stage when the epithelial organ is transformed into a lymphoid organ. No Hassall's corpuscles are present.

No lesions are found in the tissues of the scalp. The dura mater readily separates from the calvarium. The sinuses of the dura show no lesions. The subarachnoid contains a considerable amount of clear fluid in its dependent portions. The arteries of the brain are soft. The ventricles of the brain are not dilated and appear normal. A central coronal section through the brain shows no lesion.

The lumbar and thoracic part of the cervical spinal cord are removed. The meninges show no lesions. Sections of the cord show no gross lesions, except a possible softening in the anterior horns of the cervical cord.

The muscles examined were a few pieces from the sternothyroid and sternohyoid. These did not show lymphorrhagias.

Of fifty-six necropsies on cases of myasthenia gravis that have been recorded since 1901, seventeen showed hyperplasia or persistence of thymus, and ten of the fifty-six cases showed a thymic tumor. This gives a thymic involvement in nearly half of the cases. All the thymic tumors except one have been of the benign type, somewhat similar to the case reported. The authorities seem to be very general in their statements that these tumors are special forms of hyperplasia, and not true neoplasms, and therefore can best be designated as benign thymomas.

The literature on the thymus gland is neither convincing nor satisfying. The fact seems to be generally accepted that the thymus and the thyroid are interrelated, particularly from a circulatory point of view. The assumption is quite clear, too, that the thyroid is responsible for disturbances of bodily metabolism; and it seems reasonably safe to assume that the thymus is in some way responsible for like conditions. Some

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investigators believe that the thymus and the lymphatic system are coordinated in function, and that the thymus itself is probably concerned in the production of lymphocytes. It is further probable that the thymus has some corelated effect with other ductless glands; and, like other glands, the thymus may be the primary seat of the attack. This does not necessarily mean that the thymus itself is an independent organ, but that it has its influence in the ductless gland chemistry, and certain persons have disorders and hyperplasias or diseases of the thymus, while in others the same conditions cause disturbance of other glands, such as the thyroid or the adrenals. One can only theorize on the whole situation concerning the dependence of the thyroid, thymus, and adrenals, one on the other. The condition which we look on as status lymphaticus is something that we know very little about. We are led to believe from various sources that this condition may cause death under certain surroundings or circumstances. The same applies to the thymus, that there may be a status thymaticus or, as has been more aptly expressed, a status thymicolymphaticus. Some investigators do make a definite distinction between status thymicus, status lymphaticus, and status thymicolymphaticus; but this distinction can be demonstrated only after death. The group of symptoms which occur before death is not very readily differentiated.

The presence of a thymus in a person of middle life, particularly when associated with an exophthalmic goiter, is a very strong index that the thymus is the main disturbing element; and not infrequently the operator who removes the thyroid gland without recognizing the presence of a thymus hyperplasia produces a change in the circulation of the thymus which, not infrequently, is accompanied by sudden death. There is every reason to suppose, therefore, that sudden deaths in these cases of myasthenia gravis in which the enlarged thymus is present may occur, as in the case earlier recorded in this paper, and the death be a thymic death.

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The relationship between thymic disease and the central nervous system has never been established, unless it be that certain types of people who have inherent tendencies or inherent defects may be subjects of hyperplasis of the thymus, as well as other organs; and this, in turn, may have produced a change in the blood stream of the brain stem, either from indirect pressure or from substances thrown into the circulation which affect the respiratory or circulatory centers. The majority of thymic deaths in young people and children are probably due to pressure effects, yet this does not eliminate the probability of toxins in the blood stream, as well as pressure from overgrowth. This theory is based on the experiments of a few of the observers who have found nerve lesions, particularly in the gray cells, in people who have thymoma.

I am indebted to Dr. E. T. Bell of the department of pathology of the University of Minnesota Medical School for the necropsy findings, and also for his investigations in other cases of thymic tumor in myasthenia gravis, and to Dr. F. P. Moersch for his observations and assistance.

ABSTRACT OF DISCUSSION

Dr. Walter B. Swift, Boston: It has been held by Brown of Brooklyn that thymus enlargement is an etiologic factor in stammering. I should like to ask Dr. Jones if any of the cases he knows about have stammering connected with this lesion.

Dr. R. Foster Kennedy, New York: In my first two years at the National Hospital I did not see any case of myasthenia gravis. Later we had nine cases inside of eighteen months. Seven of these patients died; we succeeded in getting six post-mortem examinations. Of the six cases, five had thymic persistence. In practically all of these the thymus was as large as my hand and was as thin as tissue paper, comprising a thin sheet of glandular tissue which spread down over the whole of the upper part of the pericardium. While it was not clear to us a half dozen years or so ago what the connection between the clinical disease and these extraordinary pathologic findings might be, it did occur to our dull intelligence that it was more than a coincidence, that there must be here some relation between cause and effect. I have seen only one case of myasthenia gravis in a woman over 25. They were nearly all young women; the men patients were always in middle life. Sir William Gowers published a group

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of cases of three men as I remember, which he calls pseudo-myasthenia gravis, as a result of gasoline poisoning, or as he called it, petrol poisoning.

Dr. C. Eugene Riggs, St. Paul: I have under my care at the present time a case of myasthenia gravis in a woman who is over 25 and under 30.

Dr. George A. Moleen, Denver: A case came under my observation as a dysentery and in Colorado when we find dysenteries emaciation and marked prostration one of the first assumptions is "tuberculosis." On inquiry it was learned that it was not lung trouble for which this patient came West, but a muscular weakness. He had been examined in Chicago by a very good man some twenty-four years before and the condition was then pronounced a progressive muscular atrophy; six years later it was called myasthenia gravis. The myasthenia was evident, and it occurred in epochs. The acute attacks occurred in periods of two or two and one half years, with all the bulbar and ocular symptoms, then he would gradually recover, gaining in strength. But since the last two or three attacks prior to observation he had not recovered completely. Examination revealed tenderness under the right lower costal margin at that time, and very fortunately segregation of the urine revealed pus coming from the right kidney. Exploratory operation revealed a large perinephritic abscess which compressed the right adrenal against the lower surface of the liver on that side. Evacuation and drainage of that caused prompt return of his power. I followed the case for about six months after that, when he returned to Kansas, and took up his occupation. The case was distinctly myasthenic in type, and at that time was reported as a case of Addison's disease from a perinephritic abscess.

Dr. William Alex. Jones, Minneapolis: In the literature on myasthenia gravis I found nothing relating to speech defects, particularly stammering. I have seen but few cases of what I believed were true myasthenia gravis, and the two or three I have in mind at the present time have manifested no marked change, and are still living fairly comfortable lives. The tumor in the case I reported was about the size of a large Damask plum, and naturally occupied a considerable space in the mediastinal region.

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CONJUGAL PARESIS

REPORT OF A CASE

H. H. DRYSDALE, M.D.
CLEVELAND

Paresis (dementia paralytica, general paralysis of the insane) is a progressive, chronic and incurable malady possessing a highly characteristic symptomatology which concerns the motor and psychic functions. It eventually annihilates intelligence, disrupts the personality and chooses for its victims vigorously constituted individuals who have not previously been afflicted with mental disease. The sole cause is syphilis.

At the onset, the alterations in the psychic life may occur so insidiously as to be practically unnoticed by friends and relatives. A skilled observer, however, even in the absence of physical manifestations, will readily interpret such early signs as failing attention, sluggishness of thought, dampened perceptive acuity, tiresome reiteration, unmotivated gaiety, erratic conduct, etc., as forerunners of this disastrous disease.

In not a few cases the first warning may appear with the ferocity and unexpectedness of an earthquake—an actual mental cataclysm. When the disease is ushered in so acutely, the patient is apt to run amuck and give way to violent impulsions. Often a history of recent alcoholism complicates the situation. A serologic investigation of the blood and spinal fluid will, of course, make the differentiation.

The disease, irrespective of the mode of onset, pursues a more or less irregular course which culminates eventually in a state of permanent dementia—that final wreckage of mind which permits of no opportunity for salvage.

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In early years, paresis was pictured as an organic brain disease which had for its early manifestations grandiose ideas and extravagance of conduct. With the advent of the Wassermann era and the newly acquired knowledge and improved diagnostic methods which resulted, it became evident that many cases were improperly classified because the individual had not at some time exhibited symptoms of exaltation and grandeur. Today our interpretation of the malady has greatly changed, and it is now customary to recognize three clinical types, namely, the exalted, the hypochondriac and the demential. The exalted and hypochondriac forms are encountered more frequently among the professional and cultured classes; the demential type appears to predominate in women and among those of the poorer classes.

The course of the disease, while progressive, is subject to remissions which have always proved deceptive to the unknowing. These periods of arrest of symptoms may last for months, and in a few cases for several years. Many patients during these "temporary respites" cherish the belief that a genuine convalescence is at hand, and are often permitted to return to business and look after their personal affairs. This, at best, is always a dangerous procedure, as remissions may end abruptly; and if the unfortunate victim is not properly safeguarded, he may dissipate his funds or indulge in conduct which might prove troublesome and costly.

Leading serologists are quite agreed that paresis continues to be a hopeless and incurable affliction. Long continued investigations have disclosed the interesting information that the central nervous system, in these cases, is structurally damaged soon after the invasion of syphilis, and usually years before a physician is consulted. If this is true, the prospect of obtaining a cure for this tragic disturbance is certainly dark. Yet there are some among us who, in spite of the best information on the subject, have become "specific therapeutic enthusiasts," so to speak, and are subjecting

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well established cases of paresis, under the guise of cerebral syphilis, to intensive salvarsan therapy, and are interpreting ordinary remissions as evidence of their curative skill. Apparently these "alleged" cures are not long lived, as it is becoming quite a commonplace experience to examine patients in an advanced stage of the disease who had just previously passed through an ordinary remission following a course of intravenous or intraspinal medication.

Convulsive seizures occur in fully 60 per cent. of general paralytics. It may happen that the symptoms hitherto slight may become greatly intensified after a convulsion. In other instances, the subsidence of a seizure may mark the beginning of a remission.

The disease confines itself largely to the male sex in the proportion of about 9: 1, and makes its appearance usually when the individual is in his prime. The average age of onset is 40 years, and the period of time that intervenes between the infection of syphilis and the development of paresis ranges from two to twenty-five years. Not more than 50 per cent. of the patients admit syphilis, or are aware of having contracted it. Ten per cent. of patients admitted to state hospitals are afflicted with dementia paralytica.

Hereditary syphilis may also lay the foundation for paresis, and it is apparently the usual cause of the infantile or juvenile forms of the disease. Reports of over 100 such cases have been collected.

Fortunately not every syphilitic becomes a paretic. In fact there are in some countries much syphilis but little paresis. In Algiers, for instance, where fully 60 per cent. of the natives are syphilitic, progressive dementia is a rarity. The disease, therefore, requires other elements to bring out its development, and to cover this point, von Krafft-Ebing coined the now famous phrase "civilization and syphilization." Perhaps it may prove, as in the case of malaria, that certain strains or species of the micro-organisms are the true cause of the disease.

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Paresis has been observed in a case of a mother and child (Hoch) and in brothers (Collins, Oppenheim). The case of two brothers reported by Collins of New York is especially interesting, as the two boys attended college at the same time, and both have been suspected of contracting syphilis from the same source. Husband and wife may also become afflicted, and it is this phase of the disease which constitutes the subject of my thesis.

If the literature on the subject is a criterion, paresis appearing during the lifetime of both husband and wife is an extremely rare condition.

The "Index Catalogue of the Library of the Surgeon-General's Office, United States Army," makes no direct reference to any cases which may have occurred in the United States, but it does have a record of a few cases which have been reported in foreign periodicals. These, briefly, are as follows:

CASES IN THE LITERATURE

Laignel-Lavastine and Mercier presented the following case to the Society of Psychiatry in Paris, Nov. 21, 1912:

The wife, aged 39, an actress, had impairment of memory, megalomania, dementia, tremors of hands and lips, dysarthria and fixed pupils. Serologic examination disclosed a positive Wassermann reaction on the spinal fluid with increase of albumin and lymphocytosis.

The husband, aged 27, had less marked symptoms. Memory was not impaired, but there was a decided change in personality. Reflexes were responsive except the pupils, which were contracted and immovable. Dysarthria prevailed, and leukoplasia of the corners of the mouth was noted. The spinal fluid showed an increased albumin content, and the Wassermann reaction was positive.

These authors express the view that conjugal paresis is not altogether uncommon, but that few cases are reported because the symptoms do not develop in the husband and wife at the same time.

Ferenczi[1] publishes the report of a case of dementia paralytica in a man and cerebral syphilis in the wife. Both denied having ever contracted syphilis, and no clinical lesions were found in either patient.

1. Ferenczi, A.: Budapest. Konijl Aerzte-Gesellsc., 1903, xvi.

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The husband, aged 44, presented pronounced dementia, incoherency of speech and action, exaltation, rhombergism, and exaggerated deep reflexes.

The wife, aged 44, suffered with paralysis of the motor oculi nerve of the left eye and paresis of the facial musculature. The case was reported prior to the Wassermann era, and the author inferred that because the wife was syphilitic the husband also must have contracted the disease.

Garnier and Sautenoise[2] state that the object of their communication is to point out that syphilis is the cause of paresis, and they cite a case of a man and wife. The husband was syphilitic for twelve years and the wife for ten years. Both developed paresis and both were the offspring of psychopathic progenitors.

Sipocz[3] reports three cases of conjugal paresis and one of taboparesis occurring during his eleven years' service in the psychopathic clinic in Budapest.

Junius and Arndt[4] have collected among several thousand cases of paresis and tabes in the Anshalt-Dalldorf, during a period of twenty-five years, thirty-eight cases among man and wife. In seven cases, six men had paresis and one wife tabes, in one case, vice versa. One man was married twice. He too was a paretic. With his first wife he had one daughter who developed paresis at the age of 24. The wife finally became tabetic. The second wife was delivered of two stillborn children, and she also developed tabes.

AUTHOR'S CASE

The case which I report concerns a man and wife, both of whom are living and inmates of the same institution. Both are afflicted with paresis, which now is in the terminal or advanced stage.

THE HUSBAND

G. L., German, aged 40, a paper ruler by occupation, was first examined, Sept. 1, 1913, in consultation with his physician, Dr. John Neuberger. The patient was a man of moderate stature but poorly nourished, and was somewhat emaciated. His ancestral history proved negative so far as his present condition was concerned. No inherited psychopathic taint was disclosed. His father died of pulmonary tuberculosis at the age of 62. His mother and one brother died of organic heart disease, the former at the age of 52 and the latter at the age of 43. Two brothers and one sister are alive and well. The patient was the youngest of a family of five children. As a lad he was healthy and robust.

2. Garnier, S., and Sautenoise, A.: Reflections on a Case of Conjugal Paresis of Syphilitic Origin, Arch. neurol., 1905, xix, 110.
3. Sipocz, G.: Orvosi hetil., 1908.
4. Junius, Paul, and Arndt, Max: Monatschr. f. Psychiat. u. Neurol., 1908, xxiv, 10.

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He attended public school until he was 15. Then he went to work as a book binder and later as a paper ruler. He was a hard working, thrifty and industrious man and was considered healthy and strong. At the age of 27 he married.

In July, 1913, his wife noticed an unaccountable change in his temperament. In health he was reserved but most indulgent, affectionate and thoughtful. Now he was hateful, indifferent, and wished to be left alone. The condition was regarded as a "nervous breakdown" by his relatives. Sept. 1, 1913, he was obliged to give up his work, being no longer able to apply himself, and making many mistakes. His employers urged him to take a prolonged rest. This seemed to distress him, and he became quite agitated and disturbed. For hours he would hold his head in his hands and constantly bemoan his fate. He could not sleep; he walked the floor at night, and finally the wife came to realize the seriousness of the situation.

The physical examination disclosed total abolition of the patellar reflexes, tremors of the closed eyelids and separated fingers, fixed and contracted pupils, dysarthria, instability of gait and moderate emaciation. Mentally he was confused and distinctly apprehensive. Many of his answers were incoherent, and he was suspicious of his family. His wife's well intentioned solicitations were angrily repulsed, and he insisted that she leave the room. During her absence he voluntarily told us of having contracted syphilis in 1898. He consulted a physician, and soon thereafter considered himself cured, as all symptoms had rapidly disappeared. Two years afterward, 1900, he married, and finally came face to face with the cold truth that a cure was most unlikely, and that there was great danger of his having transmitted the disease to his wife and child. He further stated that he had carried his "secret" single handed during all these years, and had been hoping against hope that no harm had resulted from his folly and indiscretion.

A provisional diagnosis of dementia paralytica was made, and a specimen of his blood obtained for serologic investigation. Before this was completed, the patient became quite disturbed and boisterously incoherent. He could not be quieted, and assumed threatening attitudes toward the members of his immediate family. Sept. 4, 1913, it was necessary to arrange for his commitment to the Cleveland State Hospital. The result of the Wassermann test was finally reported as positive, four plus.

Since his admission to the institution the disease has pursued a progressive course with few slight remissions. At various times he has suffered with convulsive seizures, some of which were quite severe and prolonged. He is now markedly demented, stupid, untidy, resistive and partially bedridden.

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The diagnosis is dementia paralytica (paresis). The Wassermann reaction is: blood, plus four; spinal fluid, plus four. The cell count is 28.

THE WIFE

H. L., a native of Ohio, aged 36, consulted me a short time following her husband's adjudication. Both her grandparents died of senility Her father died of a complication of diseases, aged 50. One sister died of chorea, aged 30. Her mother and one sister are living and well. All the members of the family, including herself, were unmistakably neurotic. She suffered the usual diseases of childhood but developed normally. At school she was active and alert, but not especially vigorous or robust. Menstruation first appeared at the age of 13 and has since been normal. At the age of 23 she married. The family life was all that could be desired, and the man was at all times a devoted husband and father. She had known him from early childhood, and had every reason to believe that his habits were exemplary; in no manner was he given to excesses. The patient further informed me that she suffered a miscarriage shortly after her marriage which necessitated a curettement of the uterus. An uneventful recovery followed.

At this point I suggested to her the advisability of submitting to a thorough examination, but this she deemed wholly useless as her health was good. It was then explained that her husband was afflicted with a specific disease, and it was most essential to determine whether or not the disease had been transmitted to her. She refused to consent, and left the office apparently under the impression that I was mistaken in considering her husband's condition incurable. It was also evident that she questioned the cause of his invalidism.

I saw nothing more of this woman until May, 1915, when she returned complaining of nervousness, insomnia and numbness in both lower extremities. She had been visiting her husband regularly at the hospital, and this had greatly depressed her. His condition had grown gradually worse, and he was now a patient in the "infirm" ward. A specimen of her blood was secured for serologic investigation, and this disclosed a positive reaction for syphilis. Treatment, however, was still refused; in fact, she assumed an attitude of total indifference, and declared that she no longer cared what became of her, as there was nothing left to live for.

Two months afterward, July, 1915, I was summoned to her home and found her quite excitable, erotic and rambling in her talk. The pupils were contracted and fixed, the left patellar reflex abolished, the gait quite ataxic, and considerable dysarthria prevailed. She had no recollection of what had transpired the day before, and seemingly had no insight into her condition. Specific medication was pushed, and in a

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short while all mental manifestations abated and she was quite herself again. Her regular visits to her husband were resumed, and she invariably insisted on making the journey alone.

From this time on her conduct was noticeably irregular, and she became ill at ease with those who were near and dear to her. She neglected her daughter, avoided meeting people, and lost much of her former pride and neatness of appearance. Oct. 14, 1915, I received a message stating that the patient was found wandering about the hospital grounds, and requested that the family be notified. Arrangements were made for her return to the home, where I saw her the following morning. I hardly knew her when I called, as she had lost much in weight, and was profoundly confused and incoherent. During the afternoon she became quite maniacal, and the relatives, finding themselves unable to cope with the situation, took the necessary steps for her commitment to the Cleveland State Hospital.

According to the hospital records the progress of the disease has been exceedingly rapid, and at present she is almost as deeply demented as her husband who was admitted two years previously.

The diagnosis is dementia paralytica. The Wassermann reaction is: blood, four plus; spinal fluid, four plus. The cell count is 30.

THE CHILD

In November, 1915, I had an opportunity of examining the daughter, who was then 8 years of age. She was of slender stature and poorly nourished. She told me that she felt well and was progressing nicely at school. Her aunt, who accompanied her, intimated that she was subject to "colds" and had periods when her appetite was poor. She had never been seriously ill, however, and had exhibited no signs of nervousness.

The clinical examination was practically negative, and no objective neurologic manifestations were elicited. Her mentation was normal, her memory was faithful, her attention good, and her ideas were expressed coherently and connectedly. Temperamentally she was quiet and reserved. The special senses were intact and the eyegrounds were negative. A specimen of her blood was submitted to a Wassermann test, and this was found to be positive for syphilis.

The condition of the child has been made known to the relatives, and the responsibility of her future care now rests with them.

COMMENT

This case presents some very interesting features. The father and mother are now hopeless invalids as the result of the ravages of syphilis. Their only daughter

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has inherited the disease, and no one can prophesy what her future health will be. The husband contracted syphilis two years prior to his marriage. The symptoms were slight and quickly subsided after a brief period of treatment. Eighteen years of syphilis preceded the development of paresis in his case.

Unquestionably he infected his wife, but just when no one can say. She did, however, become a paretic two years after her husband, who was syphilitic two years before they were married. It would be interesting in this connection to know if the number of years of syphilis preceding the appearance of paresis was the same in husband and wife.

The disease in both cases was of the demential type, and at no time during my observation were there any signs of exaltation or grandeur.

According to the literature, cases of conjugal paresis are rare; but it has seemed to me that members of this section whose experience has been more extensive than my own must surely have encountered conditions of this sort.

I am indebted to Dr. A. G. Hyde, superintendent of the Cleveland State Hospital, for the clinical notes of this husband and wife since they came under his care and treatment.

Rose Building.

ABSTRACT OF DISCUSSION

DR. JULIUS GRINKER, Chicago: This report brings up a number of reflections on the subject of syphilis and parasyphilis. Many of the ideas in connection with this subject are still hypothetical and in need of further proof. However, clinical reports such as this one seem to throw some light on the problem. For instance, why should the wife contract general paresis and not tabes? It appears as though there might be a special strain of these spirochetes which produce a certain nervous disorder of the tertiary or of the quarternary, or, as it is now called, the parenchymatous type of syphilis of the nervous system. This fact was very strongly brought to my mind when in 1903 I had occasion to study an entire family of syphilitics, most of whom, with one exception, had developed parasyphilis; this exception was the father, who died too young to develop any form of nerve syphilis and whom I had not examined.

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The case was this: A man who had contracted syphilis married a woman who had previously been married and as a result of her first marriage had had a healthy son, who later had a healthy family. The first result of this second marriage was the birth of a boy, who early developed normally, attended college, and later entered a countinghouse, doing work satisfactorily. At about 19 years of age he developed juvenile paresis and died. The second child, a daughter, had all the evidences of hereditary syphilis on her body, snuffles and Hutchinson's teeth, but went on to womanhood and then developed an attack of right-sided hemiplegia with aphasia and in addition was the subject of a gummatous meningitis, as I determined on postmortem examination. The third offspring, a boy, developed symptoms of tabes early, with incontinence of urine and the peculiar difficulties that go with hereditary juvenile tabes, until at 19 there appeared an optic atrophy, causing total blindness, but very little ataxia. So slight was the ataxia that the patient could easily find his way through Chicago's busy thoroughfares, having made his living by peddling newspapers. The mother developed optic atrophy and tabes. There is no doubt that all had contracted the disease from the father.

It seems to me that the family tendency may have less to do with the variety syphilis will assume than the fact that there are different strains of one and the same organism. In consequence, we are beginning to believe that there are several strains of spirochetes, and the case reported by Dr. Drysdale would tend to support that theory. Referring to our new treatment of syphilis and whether intravenous salvarsan injections or intracranial injections are preferable to the old method of administering mercury and iodids, only time and more experience will decide.

Dr. John Matthew Pulliam, Fort Wayne, Ind: I have under observation at present a man who contracted syphilis twenty years ago. He is not married, but has had relations with a woman and presumably they have a child. This man is paretic; he is now undergoing a remission. The woman is paretic, and the daughter, now 17 years old, is paretic. The mother thinks the child is by this man, but is not certain. In regard to the spirochetes, I do not know that it is a question of strains. Tuberculosis manifests itself in many different forms: tuberculosis of the lungs, of the glands, and of the bones. So why could not syphilis affect the brain in a certain individual, in another individual of that family attack the cord, and so on down the line? We could have any part of the body affected and perhaps at the point of least resistance. I think this idea would be more logical than to say that there are different strains of the particular organism. I presume there are, and yet the disease might attack a point of least resistance where the conditions were more favorable to the spirochetes than in any other locality.

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DR. LAWSON G. LOWREY, Hathorne, Mass.: We have had in the state hospital three members of one family, father, mother and daughter. The mother developed paresis first, then the father, and then the daughter came to us, not for paresis or any syphilitic disorder, but because of what appeared to be a state of manic depressive insanity. She, however, had a positive blood Wassermann. The mother eventually died and necropsy substantiated the diagnosis of paresis. The father and daughter are still in hospitals, but have been transferred. The father is of excitable type, and it is rather common that people of this class live longer than the more quiet ones. As to whether or not the nervous system is the locus of minor resistance, or whether different strains of spirochetes operate to produce paresis, there is evidence in both directions. It is a problem that must be solved experimentally, one on which a considerable amount of experimental work is now being done, and I believe that at the end of three years we shall know more about the subject than we do today.

DR. C. C. BURLINGAME, South Manchester, Conn.: In view of the work reported by Reasoner, it would seem that there is little doubt as to the existence of distinct strains of the organism. Dr. Reasoner took some twenty strains of the organism and experimentally produced syphilis in rabbits. He found, say, that Strain No. 5 produced a certain lesion of the eye; Strain No. 10, no matter how frequently repeated, never produced those symptoms of the eye. In brief, he has taken certain strains of the spirochetes and produced certain definite lesions which could not be produced with other strains of the organism.

DR. DAVID S. BOOTH, St. Louis: There came under my observation a family consisting of a wife and her two husbands, which I wish to report briefly since I do not know how much may have been sequence and how much coincidence. I was brought into the family to examine the second husband, making a diagnosis of paresis, when I learned that the first husband had been confined for the same disease and had committed suicide in the insane asylum. The second husband was committed to the asylum, and several years after, the wife was also committed to the hospital in the same condition. And one of these two committed suicide; I do not remember which.

DR. WILLIAM RAVINE, Cincinnati: In 1912 I saw a woman brought into the Anton Clinic at Halle a. S., in a state of depression with the physical and serologic findings of paresis. About 15 years before her admission to the clinic her husband was brought in with the diagnosis of paresis; he was sent to the state insane asylum and died of paresis. This woman married two years after the death of her first husband, and eight years later her second husband developed signs of

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paresis, was brought into the clinic, and subsequently sent to the asylum. He was still alive on her admission. Permission was obtained for the performance of brain puncture of husband and wife, and the typical lesions were produced in the eyes and testes of rabbits. In a recent study of about 75 children of paretics, I was astonished to find that but one child showed evidences of hereditary syphilis, and but two gave positive Wassermann reactions in the blood; spinal punctures were not made.

Dr. W. F. Lorenz, Mendota, Wis.: We make it a practice to examine relatives of paretics in the Mendota and Northern Hospitals. A large number of children have been examined, and less than five per cent. have shown a positive Wassermann. As a rule, they appear normal and bright, and, in fact, most of them are healthy and good-looking.

Dr. Charles R. Ball, St. Paul: I recently had under my care the daughter of a paretic, age 17. The Wassermann test was negative. On two occasions the luetin test has been positive. This young woman is extremely nervous and erotic and seems absolutely devoid of any moral sense or responsibility so that at the present time she has to be watched constantly. She is extremely immoral and does not hesitate to steal or forge checks, and when confronted with her misdeeds manifests no sense of shame or remorse. There is no doubt but her moral void is an effect of the syphilis of her father. She might be properly classified under the cerebral palsies. The difference is simply one of localization; in this case the moral zone was damaged instead of the motor.

Dr. George H. Moody, San Antonio, Tex.: For many years I have been observing children of paretics rather closely and have made Wassermanns on some of them. This observation leads me to believe that children of syphilitic parents whose syphilis has existed for a long time are much more likely to appear normal and not so prone to present degenerative signs of hereditary syphilis as are those children whose parents have recently contracted syphilis, indicating that if an individual has had syphilis perhaps the time elapsing since contraction of the disease is the most important element in modifying the severity of the infection.

Dr. S. G. Burnett, Kansas City: I have as patients four brothers, each having paresis, the ages being 26, 30, 36 and 40 years respectively. Each gave an individual history of having contracted syphilis. The son of the oldest, at the age of 17, contracted syphilis and five years later developed paresis, the five patients contracting syphilis from different sources and all having paresis. It is hardly likely that the one strain of infection would be contracted by all these individuals. So the question is whether we have a special strain producing the same condition in different individuals

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derived from different sources, or whether we have the infection attacking the tissue which is most liable to be involved in the process.

Dr. Robert Henry Haskell, Ionia, Mich.: At the State Psychopathic Hospital, Ann Arbor, for several years we took the blood reaction of wives or husbands of paretics and likewise of the offspring, if any. While we found that about 40 per cent. of the conjugal mates of paretics had a positive Wassermann, the percentage of positive reactions in children was very small, less than 10 per cent. I believe. In fact, these children were, as a rule, good-looking and bright. As you go through the family history in these cases you find that the number of deaths of children in early life is excessive, so that while the census for the state of Michigan shows about three living children in each family, in the families of eighty-four paretics we found on an average less than one living child in the family after an average married life of eleven years. It seems to me that why most of the children of paretics fail to show a positive reaction is that the actually infected ones have died young. Hauptmann lays great emphasis on the differences between actual hereditary infection with the spirochete and syphilitic intoxication of the germ plasm, a distinction which I believe we must keep clearly before us.

Dr. George A. Moleen, Denver: About seven or eight years ago a woman was taken with apoplectiform seizures, in which there was introduced a type of paresis of the dementia class with confusion and other symptoms of the condition, which progressed, and in about four and one-half to five years she died in an institution. At about the time of her death the husband began to show definite signs of a paretic type with loss of reflexes and some incoordination, however, with the tremor and then with the apoplectiform seizures, of which he has had two, without suffering a dementia, and only since the apoplectiform seizures has he had some confusion. Neither case, in fact, showed optic disturbances in the form of atrophy, but both presented the pupillary phenomena. The child of this conjugal relationship was a definite, simple-minded imbecile of rather a higher grade type. He presented the evidences and the stigmata of degeneration. With a little assistance he would probably be able to make his way in the world and help himself, but I doubt very much whether he will make any progress alone.

With reference to the strains: Many theories have been advanced, from the neurotropic theory of the French, and the symbiotic view of the influence of other organisms on the spirochete, as well as a selective affinity on the part of some nervous systems for the neurotic influence. Yet, as has been said, it will take time and judgment before we can arrive at definite conclusions as to whether or not there

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are such strains, even in spite of what would seem to be very good evidence at the present time.

DR. HARRY H. DRYSDALE, Cleveland: The condition does not seem to be as uncommon as the literature on the subject would indicate. It is to be regretted that these cases have not been reported. In my experience also, in the examination of children of paretics not more than one in ten gives a positive Wassermann. It is surprising that in examining children of parents afflicted with some form of syphilis, the Wassermannn test is so frequently negative. I am extremely glad that I have reported this case as it has brought forth, in discussion, some very interesting and valuable points.

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HEREDITARY SYPHILIS AS A CAUSE OF CHRONIC INVALIDISM

AND THE DIAGNOSIS BY INTENSIVE FAMILIAL STUDY

HENRY FARNUM STOLL, M.D.
Visiting Physician to the Home for Crippled Children, Newington; Assistant Physician, Hartford Hospital; Physician-in-Chief, Syphilis Clinic, Hartford Dispensary
HARTFORD, CONN.

There is scarcely a symptom to which man is heir that may not be caused by the *Spirochaeta pallida*.

With the development of delicate laboratory tests the field is ever widening, and the syphilitic yardstick has been applied to practically every syndrome, with the result that we have been forced to change many of our ideas, and the end is not yet.

For many years certain clinicians have realized that syphilis is a disease of the family rather than of the individual; but it was not until the advent of the Wassermann test that it was appreciated how extensive prenatal infections are and that an apparently healthy child may harbor the organism.

Time causes certain changes in the spirochete in vitro; for example, the organism from a primary sore produces a lesion in a rabbit's testicle in from two to three weeks, while that obtained from general paresis requires three or four times as long to develop.

In like manner the symptoms occurring from twenty to thirty years after infection — especially prenatal infection—are often insidious of onset, vague and indefinite in their development, and quite frequently unaccompanied by physical signs. To obscure the condition further, the Wassermann test, so valuable in early syphilis, is only rarely positive in adults with hereditary syphilis.

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For several years I have been interested in the study of syphilitic families; their symptoms have been analyzed, their physical signs tabulated whenever possible, and Wassermann tests made on many. I have been greatly assisted in this work by Miss Barry of the Hartford Visiting Nurses Association and Miss Goldberg at the Hartford Dispensary.

As a result of a more or less complete study of approximately 100 families, it has seemed to me that if one groups together all the symptoms and physical signs of all members of the family, certain syndromes will often be found that are quite suggestive of what might be termed familial syphilis.

The individual with tuberculosis usually presents a combination of symptoms and physical signs that are more or less typical of the disease. In "late" hereditary syphilis, however, the patient may present no sign of syphilis and but a single obscure symptom which may be so distressing that a more or less complete invalidism results. But suspicious symptoms and perhaps conclusive physical signs may be found in other members of the family, and in this way a wholly indefinite and meaningless symptom, like one piece of a picture puzzle, may become highly significant when fitted in to its right place in the family syndrome.

I judge that the diagnosis of hereditary syphilis by intensive familial study is not generally practiced, as no mention was made of it in four symposiums which I recently attended where many well known syphilogists discussed the various problems of syphilis.

Family histories as commonly taken are entirely worthless in deciding for or against the possibility of familial syphilis. It takes a good deal of time to elicit all the facts; but not infrequently the information thus gained furnishes the only means of clearing up an obscure case.

Within a few days I examined a man whose father was stated to have died as the result of an accident. Inquiry developed that the accident was a gunshot

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wound through the mouth from which he made a good recovery, living many years thereafter and leading an active business life. About ten years before he died he developed some spinal trouble, which was attributed to his previous injury. He lost the use of his legs and got about only in a wheel-chair. This suggests that he may have had tabes, which would explain the high narrow palate I observed in his son and quite possibly the cerebral endarteritis from which he was suffering.

Before attempting to work out various syndromes of familial syphilis, one must first appreciate what diseases in a parent are indicative or highly suggestive of syphilis. We are justified in assuming syphilis when a parent has had tabes or paresis; as probable syphilis a history of aneurysm or aortic disease, and death from apoplexy and sudden heart failure before 50; as possibly syphilitic, cardiovascular renal deaths up to 60 or perhaps beyond. I also believe we are justified in at least suspecting syphilis in the presence of severe chronic headaches not relieved by glasses. The parents may be living and said to be well; but a paralysis of the third or sixth nerves, insomnia, extreme nervousness or "rheumatism in the legs" should awaken our suspicions.

The second family cited illustrates the importance of minute inquiry as to the health of maternal aunts and uncles.

What is often referred to as the typical sequence of syphilitic pregnancies, namely, miscarriages, stillbirths, premature syphilitic children and finally healthy children, has in my experience been encountered only rarely. While syphilis is the most common cause of abortion, it should be realized that in certain syphilitic families all the pregnancies may result in living children. Syphilis is also a frequent cause of sterility; this seems especially liable to occur in the "weakling" group of heredosyphilitics. One should bear in mind that hereditary syphilis may have the same effect on the fetus that acquired syphilis has.

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When pregnancy results in a syphilitic, one of several things may happen:

1. The fetus dies, and abortion or stillbirth results. This is usually attributed to a uterine malposition.

2. The fetus does not die, but is expelled prematurely.

3. The child may (*a*) present obvious evidence at birth or within a few weeks, or (*b*) appear healthy for several years and then develop some syphilitic manifestations, as interstitial keratitis or a bone lesion.

4. Occasionally a healthy child is born to syphilitic parents whose disease is in a latent condition.

5. The child may exhibit certain stigmata, the result of protoplasmic disturbances, even when no infection occurs. I have had as patients two men with acquired syphilis with high narrow palates who were the sons of syphilitics. These dystrophies or alterations of the normal development of the germ plasm may be accompanied by a real congenital syphilitic infection. The common dystrophies are the high narrow palate, hare lip, scaphoid scapulae, short arms, hypoplastic teeth, etc.

6. We should suspect the possibility of hereditary syphilis in individuals whose chief characteristic is a constitutional inferiority. As children they "took" everything and were always very ill. Even when not definitely sick they were ailing and "hard to raise," yet without definite reason. Usually nervous and irritable, frequently complaining of headache, they are prone to develop tuberculosis which is apt to be fatal. Often a very thorough examination will reveal a suspicious sign, though sometimes we will find only the telltale dystrophy or symptom in a brother or sister.

It would be a very grave mistake to assume that all delicate children are heredosyphilitic. Nevertheless I am sure that too little attention has been paid to this particular disease as a cause of "weaklings."

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As illustrative cases I have purposely chosen several who were free from the usual stigmata of hereditary syphilis and who had negative Wassermanns. Most of my tests were made in our state laboratory at Middletown. A few, however, were made in reliable laboratories in New York and Philadelphia, and they too were negative.

The luetin test was positive in nearly all of the cases, but inasmuch as the specificity of this test has been seriously questioned in patients who have received potassium iodid, the results are not recorded, as nearly all had received this drug.

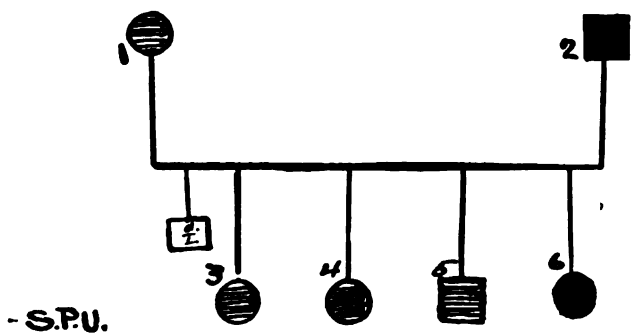

Chart 1.—Heredity chart, Family 1. In the charts, squares indicate male, and circles, female; all black, syphilis known to have been present (except Chart 5, where solid black indicates epilepsy); shaded, probable syphilis; triangle, miscarriage; double triangle, stillbirth; *d. I.*, died in infancy; *H*, headache; *TB*, tuberculosis; *A*, alcoholic; *N*, nervous; *S. P. U.*, sequence of pregnancies uncertain. In the legends, *a* indicates alive and *d*, dead. 1, *a*, alcoholic; headaches; 2, *d*, tabes dorsalis; 3, *a*, nervous; 4, *a*, nervous; severe headaches; 5, *a*, very nervous; 6, *a*, chronic headache; spondylitis.

I personally feel that an intensive familial study of the supposedly nonspecific reactions will materially diminish their number.

REPORT OF CASES

Case 1.—An unmarried woman of 26 (No. 6, Chart 1) complained of severe headaches from which she had suffered since earliest childhood. She was referred to me by Dr. Paul P. Swett, to whom she had been referred because of pain and tenderness of the upper cervical vertebrae. Dr. Charles A. McKendree, under whose care the patient was for a time at Cromwell Hall, kindly allowed me to supple-

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ment my history with his notes. She had been very strong as a child, but her headaches dated from her earliest recollection. She had always been of an excitable temperament, quick and irritable. The headaches were so severe when the patient was 15 that she was forced to leave school, and consulted a number of eminent men in Europe after failing to obtain relief in this country. Each summer for the past thirteen years she had gone abroad, as the ocean trip helped her considerably. Two years ago a well known specialist in London did an extensive operation on the interior of the nose for the relief of the headaches, but without success. A roentgenogram revealed a tooth that had failed to erupt, and with the hope of obtaining relief it was removed, but no improvement followed.

The circumstances of the patient were such that she was able to consult many specialists of renown. Several Wassermann tests had been made, but all were negative. One neurologist had recently told her he was sorry her blood was negative, as otherwise he could have given her treatment. "I know you will be disgusted," he told her, "to have me say it is just 'nerves'." An interesting fact was elicited, however. Several years ago while in Germany a physician injected some medicine into her hip, and following this treatment her headaches were very much better for a time.

Recently she has complained of paresthesia of the postcervical region; her sleep has been interfered with by nervousness, and pains in her neck which required her to frequently change her position. In the morning she is not rested. Her mother states that her condition is the result of her being a "spoiled child and growing too rapidly." Her nervousness was supposed to have been an inheritance from her mother, and her mother's nervousness was induced by the worry and strain of her husband's long illness—a highly reasonable explanation. What were the grounds for suspecting syphilis? Out of a group of 100 people she would have been the last one suspected of having hereditary syphilis, as far as her appearance went. She was a strikingly handsome woman, very tall and mentally alert. Her unusual height — she was over 6 feet — recalls to mind that Fournier, who recognized the syphilitic origin of tabes and paresis many years ago, likewise stated that gigantism and dwarfism were commonly the result of hereditary syphilis.

Dr. Swett found considerable thickening and marked restriction of lateral movement of several cervical spines, which he diagnosed as a probable specific spondylitis; otherwise the physical examination was negative.

The patient was the fourth of five children. One died in infancy. Two married sisters and a bachelor brother are very nervous. One of the sisters is exceedingly so, and also suffers severely from headaches, spending a large part of her time in various institutions taking cures. The provisional diagnosis of hereditary syphilis would probably not have been made,

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however, except for the fact that her father (No. 2, Chart 1) had died at 47 from tabes, from which he had suffered for twelve years.

Her mother (No. 1, Chart 1), who is living, formerly suffered from headaches, and is addicted to alcohol.

Notwithstanding her negative Wassermanns and inconclusive luetin reactions, she was given injections of mercuric salicylate by Dr. McKendree and subsequently by her physician in New York, with marked relief of her symptoms.

Had the father died of some acute illness or an accident before tabes had been recognized, the chief key to the problem would have been lacking; yet the extreme nervousness in the brother and sister should suggest their careful examination for some dystrophy of hereditary syphilis.

Two years ago I reported a similar case; a man, aged 36, whose father had tabes, obtained complete relief from severe headaches and many neurasthenic symptoms following salvarsan and mercury, though his Wassermann was constantly negative. A third case has recently been seen. A middle aged woman whose father had died from tabes experienced great relief from headaches of years' duration following specific treatment. She also had a negative Wassermann.

CASE 2.—A married woman of 37 (Chart 2, No. 15) complained of burning and pressure on the top of her head and of a burning at the tip of her tongue, which symptoms had been present for ten years. For many years her endurance had been poor, and often after the day's work she would have to let her hair down because the burning and pressure on the top of her head was so severe. Sometimes she would suffer from slight vertigo. Her discomfort was especially marked at the time of menstruation. For years she had been more or less disinclined to social activities because of general weakness and the above mentioned symptoms. She had been assured that they were due to "uric acid poisoning," and had been on a meat free diet for the past year. No relief, however, followed this abstinence from meat. Her only pregnancy had resulted in a miscarriage.

The patient was a large, fine looking woman presenting no stigmata whatsoever of hereditary syphilis. Physical examination was negative except that there was a slight hypertension—systolic blood pressure 140. The urine contained the smallest possible trace of albumin. The Wassermann was negative. The family history at first appeared to be entirely negative. Her mother died of cardiac dilatation following pneumonia, her father from a tumor of the throat. She has two living sisters, one is in good health, the other a semi-invalid from "too much uric acid." Two brothers are living and well, and a brother died from heart disease. Certainly a negative family history. A different picture is obtained, however, when one makes an intensive study of the brothers and sisters and of the preceding generations.

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In the first place, the sister who suffered from "uric acid poisoning" (No. 14) had a systolic blood pressure of about 260. The other sister (No. 13), who appeared well, had a systolic pressure of about 170. Both had negative Wassermanns. The latter as a child had an ulcer of the rectum which healed slowly and left a stricture. One living brother (No. 17), who was not examined, is stated to suffer from a chronic "sore" near the anus and periodically the soles of his feet "peel off." A third brother (No. 18), is apparently in good health. The brother who died suddenly at 16 of heart disease (No. 16), had never suffered from "rheumatism," which is suggestive of syphilitic myocarditis. The mother had four unmarried sisters and one brother. One sister (No. 5) was

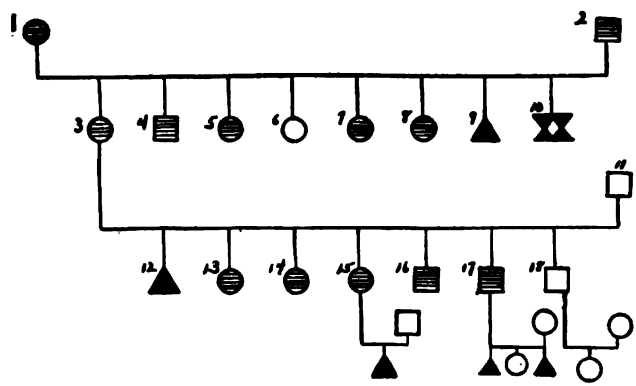

Chart 2.—Heredity chart, Family 2: 15, neurasthenic, aged 39, Wassermann reaction negative; 13, *a*, 39; hypertension; stricture of rectum as girl; Wassermann reaction negative; 14, *a*, 43; hypertension; Wassermann reaction negative; 16, died at 16 of "heart disease"; no rheumatism; 17, *a*, 37; not examined; a "sore" near anus; periodically "sheds" skin of soles; 18, *a*, 36; apparently well; Wassermann reaction negative; 3, *d*, 50, mother of preceding; cardiac dilatation following pneumonia; 4, mother's brother; died of "apoplexy" at 17; 5, sister, died at 38 of "heart disease"; no rheumatism; 6, *a*, 63; hypertension; several strokes of apoplexy; Wassermann reaction negative; 7, *a*, 60; apparently well; Wassermann reaction negative; 8, *a*, 58; was a seven months' baby; not examined; from history, probably hypertension; 10, stillborn twins; 1, maternal grandmother of 15, died at 64 of apoplexy; 2, husband of 1, died at 64 of "fatty heart."

found to have hypertension and had had several strokes of apoplexy. A second (No. 6), is apparently in good health; a third (No. 8), who was a seven months baby, was not examined, but is said to suffer a great deal from dyspnea, which is suggestive of cardiovascular disease. The fourth sister (No. 7) died at 38 of heart disease. She was unmarried and had not had rheumatism. A maternal brother (No. 4) died at the age of 17 of apoplexy. There were also one miscarriage and two stillbirths.

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In the preceding generation, that is, the grandparents of the patient with the burning tongue, the grandmother (No. 1) died at 64 of apoplexy and the grandfather (No. 2), at the same age of "fatty" heart. It is extremely suggestive that the cardiovascular disease which was present in three generations was due to syphilis.

The patient with the burning tongue and pressure on the top of her head was put on injections of mercury and potassium iodid, and promptly and completely recovered from her symptoms, which have not recurred for a period of two and one half years. Since the treatment she has become the mother of twins.

CASE 3.—An unusually alert, keen man of 30 (No. 11, Chart 3), became dazed while riding in the cars one day. On reaching home he vomited and had several severe convulsions, involving his whole body. For several days thereafter he was completely disoriented; then for a number of weeks he was listless and disinterested in his affairs, and from time

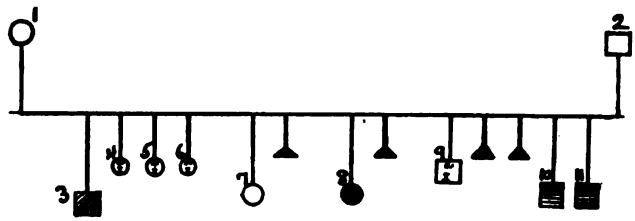

Chart 3.—Heredity chart, Family 3: 1, hypertension, died at 70 of uremia; 2, died at 52 of pneumonia; 3, hypertension; diabetes; acquired syphilis; 4, 5, 6, 9, died in infancy; 7, ♂, well; not examined; 8, ♂, 39, chorea; retarded development; Wassermann reaction + +; 10, ♂, 34, neurasthenic; visceroptosis; general pains; prematurely old; Wassermann reaction negative; 11, ♂, 30; petit mal; Wassermann reaction negative; spinal fluid negative.

to time he was exceedingly nervous. During the succeeding months he had many momentary lapses of consciousness. He would be talking with a man, and suddenly realize that this man had left and another had taken his place, but he had no recollection of the one leaving or the other coming in. Once at dinner when a plate of food was passed to him he asked "what is this for?" but instantly realized it was his dinner.

For a year prior to this illness he had been under a peculiarly severe nervous strain. Two or three times in previous years he had some sort of brief sickness apparently depending on constipation, from which he suffered. He was a slender, enteroptotic type of individual presenting no signs of hereditary syphilis. (The scapulae were not noted.) He gave a negative Wassermann at two different laboratories, and his spinal fluid was reported as negative. The Lange test was not done. Nevertheless, from an intensive study of the fam-

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ily history, I felt that the underlying cause of his attacks of petit mal was probably hereditary syphilis, and advised specific treatment.

The father died at 52 (No. 2) of pneumonia. The mother (No. 1) had hypertension for years, dying of uremia at 70.

In the first place it is significant that out of thirteen pregnancies four were miscarriages and four died in infancy. The oldest son (No. 3) escaped a prenatal infection only to acquire one later in life. The oldest daughter (No. 7) appeared to be well, but was not examined. The next oldest, an unmarried woman of 40 (No. 8), is said to have been delicate as a child. It is noteworthy that the pregnancy preceding the birth of this child resulted in a miscarriage and that three of the four succeeding pregnancies were likewise miscarriages; the fourth child died in infancy. At 12 she was very anemic. In her school work she was not up to grade, as she had difficulty in "grasping things," as she expressed it. For this reason a private tutor was provided, but she made very little progress because she was unable to concentrate her attention on what her teacher was saying. At 24 years of age she had a very severe attack of "nervous prostration." She cried much at this time, and thought people were talking about her, making unjust accusations against her character. She spent a month in bed, and ever since she has been extremely sensitive about being with people and has been more or less of a recluse. Nine years ago while on a shopping tour her legs began to tremble. She was taken home and was given a hypodermic and the twitching disappeared in about half an hour. For about six months the left side of her face would twitch; it was called "chorea."

Menstruations have been normal, but for about a year were preceded by an uncontrollable attack of "ugliness," as she described it. At these times she could not control herself, and several times she has required morphin. She is considered incapable in the household, and is not able to assume any responsibility. Her acquaintances consider her "peculiar." The family appears to be of the opinion that she is naturally disagreeable, and that if she really tried she could be like other people. She has been almost constantly under the care of a nerve specialist. The last few years she has been less nervous, though she is still far from normal. She is a tall woman, and appears to be in good health. It was not possible to make a physical examination. The Wassermann reaction was two plus (50 per cent. inhibition).

The next youngest brother, aged 34 (No. 10), has always been a weakling. I first saw him in May, 1911, at which time he complained of stomach trouble and dizziness with gas after meals, and suffered with constipation. His tongue was "parched on awakening" and while he did not belch, he had a "continuous flow of foul gas" and also "trembling in his stomach." He was also a slender enteroptotic indi-

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vidual, sallow of complexion, delicate in appearance and with all the earmarks of a neurasthenic. He was put on a diet for constipation, given olive oil injections and exercises, and he improved considerably. For a time he had some mucus in his stools accompanied by cramps, but this finally disappeared. His whole demeanor, however, was one of listlessness, and he goes about most of the time as though in a trance. He is prematurely gray, and looks careworn and old.

It did not occur to me that he might be a heredosyphilitic when I first saw him five years ago, for at that time, in common with most physicians, I thought syphilis always wore the scarlet letter. His Wassermann reaction was negative a year ago, but he was put on mixed treatment, and at the end of three weeks his older sister volunteered the information that she had "never seen such a change in any one in such a short time." She said, "He is awake in the morning at the table; he reads the paper, seems alert and complains of no aches or pains. Prior to this his only conversation had been about his ill health." She also stated that he would go to business where formerly after breakfast he would sit down and go to sleep. She seemed to think there was some possibility that he might amount to something in business, so great was his improvement. The youngest son, the patient who suffered from the attacks of petit mal has already been described. He was seen by two New York consultants, a neurologist and a syphilologist both of national repute, who saw no reason for assuming a syphilitis basis for the attacks; "Were not all the tests negative?" Quite naturally this family passed from my observation.

Case 4.—A married woman aged 35 (No. 20, Chart 4), complained of extreme nervousness. As a child she was very nervous, and would often awake screaming at night, fearing some one was in her room. Only when her father slept in her room, between her bed and the door, was she reassured. In childhood she suffered from pain in her right leg. This pain was always along the shaft of the bone, and never in the joint. For one year she had daily massage without benefit. Mud baths and hot salt rubs were also tried, but with no relief. Part of the time she was under the care of the physician to the royal family of Sweden. Tuberculosis of the bone was diagnosed, and when she was about 12 years old several operations were performed on the right leg below the knee, and also above the ankle and along the femur. She continued to have pains in her right leg, however, which were always most severe at night. For years she has slept poorly, partly because of nervousness, but largely as a result of the "rheumatism" in her leg. Walking about would often stop these pains, and nearly every night for years she has gotten up and walked the floor, often dozing off while walking, and waking as she stumbled over something. She had very poor endurance and occasional night sweats.

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Six years ago, as the result of a fall, the right knee was injured and the patella is now firmly adherent to the tibia. The patient was so nervous that it was difficult for her to give the facts of her history. She presented no signs of hereditary syphilis. Teeth, palate and scapulae were normal. The pupils also were normal; left knee jerk very active, right not obtained, owing to the deformity resulting from the injury six years ago. There were several scars along the right femur and the tibia and over the lower third of the tibia tenderness was elicited.

The history, then, was that of a very nervous child with some disease of the bones of the right leg. This child developed into an unstable, nervous woman who slept poorly, complained of weakness, pain in her right leg and occasional night sweats. This was not the history of hereditary syphilis, surely, nor did the physical examination suggest it. The family history, moreover, is chiefly remarkable in that a large

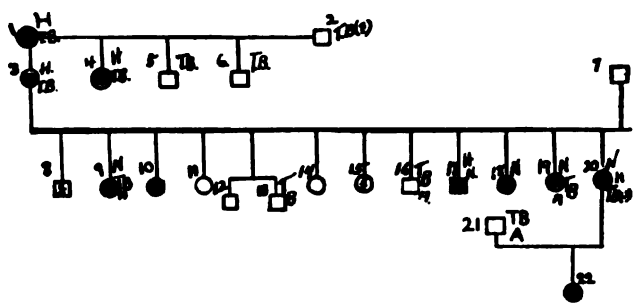

Chart 4.—Heredity chart, Family 4: 1, severe headaches; died at 60 of tuberculosis; +; 2, *d*, tuberculosis (?) "old man"; 3, severe headaches; tuberculosis at 30; died at 64; 4, headache; died of tuberculosis at 60; +; 5, 6, died of tuberculosis; 7, *d*, 60, cancer of the stomach; 8, 15, died in infancy; 9, chorea; extremely nervous; strabismus; alcohol; died at 45 of tuberculosis; 10, *a*, 50, blind several months; 11, very myopic; died at 4 of "tumor"; 12, alive, well; 13, *d*, tuberculosis; 14, *d*, "tumor of neck"; 16, *a*, tuberculosis; alcohol; 17, *a*, very nervous; "terrible headaches"; 18, *a*, very nervous; 19, very nervous; alcohol; died at 19 of tuberculosis; 20, *a*, 30; +; very nervous; headache; bone disease (tuberculous?); "rheumatism"; Wassermann + + + +; cured (?) by treatment; 21, husband of 20; Wassermann negative; 22, aged 11; interstitial keratitis; deaf; Wassermann + + + +; "cured" (?) by treatment.

number died of tuberculosis ("TB," Chart 4). Her mother, maternal grandmother, maternal aunt and two uncles, two sisters and two brothers had all suffered from tuberculosis. Questioning, however, developed other symptoms that could not be attributed to tuberculosis.

While her mother (No. 3) died from tuberculosis, she suffered for many years from such headaches that she frequently would say she could stand the cough if the headaches would only stop. The mother's sister (No. 4) also

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had severe headaches, as did the maternal (No. 1) grandmother. Among her own brothers and sisters four are exceedingly nervous (9, 17, 18, 19), one has severe headaches, one strabismus, another was blind for several months (Keratitis), another is very myopic, and three have been alcoholic since early life (Nos. 9, 16 and 19).

It is unlikely that I would have thought of hereditary syphilis in connection with this patient had it not been that her only daughter (No. 22), a large, well developed girl of 13, had been a patient of mine with interstitial keratitis and an eighth nerve involvement. This daughter and her mother had four plus Wassermanns, but the father (No. 21) gave a negative test.

The response to treatment confirmed the diagnosis. The patient received a number of injections of salvarsan and for several months took mercuric inunctions. The improvement was so prompt and so marked that she could not understand it. From a high strung woman on the verge of tears she has developed into a perfectly calm self-contained individual entirely free from any suggestion of "nerves."

Six months after the first salvarsan she said that she had not lost one night's sleep since the treatment. A month later, she stated that she was in better health than she ever had been before in her life, and what seemed most surprising was the fact that, notwithstanding several weeks of almost constant rain, she had had absolutely no rheumatism. This past winter, though she has worked in a little grocery store with scarcely any heat, she has had no recurrence of her pains.

This family illustrates in the first place how deceptive an incomplete family history may be. On the surface it appeared to contain only an unusually large number of cases of tuberculosis; but a complete study reveals certain symptoms that tuberculosis would not explain. It also illustrates the truth of Fournier's contention that hereditary syphilis presupposes to tuberculosis. But most important of all, it illustrates that the study of a child may shed some light on the ill health of a parent.

It is not known through how many generations syphilis can be transmitted. The activity of the process in the daughter with interstitial keratitis, the offspring of a heredosyphilitic mother, was manifestly such an active infection, that there seems little doubt that she could have given birth to syphilitic children. Had the child been a boy it is perhaps possible that he could

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have passed on the disease to another generation from infection by means of his semen.

During the past year three children have come under my observation because of a marked nasal catarrh. The discharge was thick and purulent, though not of offensive odor. The Wassermann was strongly positive in two. The third, the son of a syphilitic, was not tested, but he was put on inunctions of mercury with immediate and marked improvement. An equally satisfactory result was obtained in one of the other cases. The third child did not receive treatment. The following case is illustrative of what may result when such a condition is unrecognized.

CASE 5.—An unmarried girl of 18 (No. 35, Chart 5), was referred to me three years ago by a nose and throat specialist who suspected she might have a syphilitic process of the interior of the nose. She was a pretty buxom girl with excellent teeth, a normal palatal arch, and presenting absolutely no signs of hereditary syphilis. There was a purulent foul smelling discharge from the nose, which was increased in width at its base. She gave a strongly positive Wassermann. The doctor who referred her to me suspected she was an immoral girl, and I concurred with him in his opinion. It was not until over a year later that I made a special study of her own early history and that of her family. When she was 8 or 10 years of age she was a mouth breather; the condition was not improved by the application of adhesive plaster on her mouth, and finally at about the age of 15, her adenoids were removed. She still continued to have a nasal discharge, and received treatment from various specialists and clinics. She was at first told it was hay-fever and that in the fall it would probably go away. The nasal discharge increased, however, and she would have crusts form in the interior of the nose.

Shortly before I saw her a physician had told her she had probably contracted a contagious disease from a drinking cup, but the nature of the disease she was entirely ignorant of. One month previously, her nose had begun to swell. Over a year later a sister of this girl, aged 23 (No. 32), came to me complaining of transient swelling of her lips (angioneurotic edema?) and nasal catarrh. She was always "catching cold in her head." Her endurance had always been poor. Although 23 years old, she did not appear to be either mentally or physically more than 18. Except that she was a slender, frail, immature girl, her physical examination would be called negative but for her teeth. She had probably had

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hypoplasia of all six year molars; they were extremely yellow, and the lower incisors were yellow at the tips. The Wassermann was three plus.

Several months later another sister, aged 22 (No. 33), consulted me because of sudden "cramps" in the calves of the legs (intermittent claudication?). Following an illness at the age of 12, said to have been typhoid, she had ulcers on her legs. She was exceedingly nervous, and all of her life had suffered more or less from headaches chiefly across the forehead and invariably worse at her menstruation periods. Her Wassermann was strongly positive.

A third sister, aged 19 (No. 34), suffered from epilepsy. She, too, had been subjected to severe headaches, and in childhood had suffered from severe pains in her legs, referred

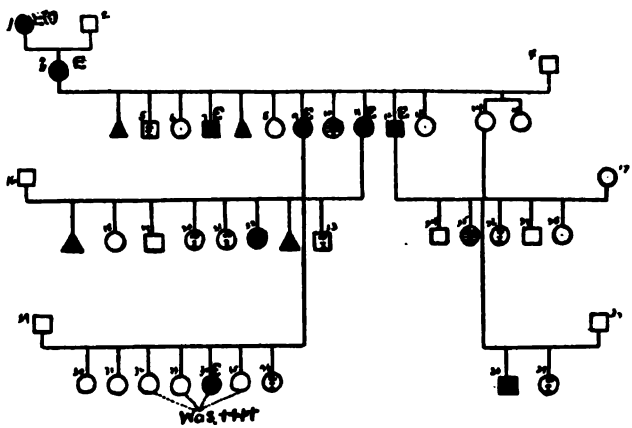

Chart 5.—Heredity chart, Family 5: 1, *d*, 84, probably epileptic; 2, *d*, 80; 3, *a*, epileptic; 4, *d*, 62, arteriosclerosis; 6, *a*, well, 60; 7, epileptic, *d*, 40, heart and kidney disease; 8, *a*, well, 50; 9, *a*, epileptic; married; 29; a drunkard and suicide; positive Wassermann reactions from their children, 32, 33, 34 and 35, and 34 is epileptic; 10, teeth at 1 week; dropsy; died at seventh year; 11, *a*, epileptic; had two miscarriages; three children died in infancy; a daughter, (22), arrested development; 12, *a*, epileptic; Wassermann reaction negative; 13, *d*, Bright's disease; aged 23; 14, *a*, said to be well; her son, 38 has a very large head and is mentally deficient; 15, *d*, 21, cause (?); 5, 20, 23, 25, 26, 36, 39 died in infancy; 24, *a*, frail, Wassermann reaction negative; 25, "blue baby"; 28, very frail.

to as "growing pains." She had always been nervous. The Wassermann test showed complete inhibition. Following the use of mercury inunctions, the nervousness was notably less.

This family, then, presented four girls, one of whom had rhinitis, three were very nervous, one was an epileptic, one gave evidence of an arrest of development both physically and mentally, and two had headaches.

As the father was a drunkard and suicide and the mother an epileptic, the so-called neuropathic heredity would explain

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all of the symptoms, save the nasal lesion, were it not for the positive Wassermanns. It is quite possible that the mother contracted syphilis from her husband. There is considerable, on the other hand, to suggest that the mother may have been heredosyphilitic.

One maternal aunt (No. 11) and two maternal uncles (Nos. 7 and 12) suffered from epilepsy, as well as the maternal grandmother (No. 3), who is still living at the age of 80. Her health otherwise is excellent, and she is a very active woman. Her mother (No. 1) lived to the age of 84, and is also stated to have suffered from "fits." One maternal aunt, (No. 11), and uncle, (No. 12), and the grandmother gave negative Wassermann tests.

It was not possible to make a complete study of the children of the other members of the family. A daughter (No. 22) of one of the epileptic aunts, however, was immature in appearance and had abnormal teeth. This aunt had also had two miscarriages and lost four children in infancy or childhood. Another sister (No. 14) has one living child of 6 years who has a very large head and is mentally deficient. Another of the mother's third sisters (No. 10) had teeth at one week and died of dropsy at 7 years of age. Two sons (Nos. 7 and 13) died from kidney disease.

Care must be exercised in differentiating between what might be called incidental syphilis which merits consideration but no therapy, at least for the moment, and what we may designate as pertinent syphilis.

As an example of the former, mention might be made of a case of pneumonia recently seen in consultation. The patient, a young woman, had a large bulging forehead. Two sisters had similar foreheads. The mother was prematurely gray and very nervous. The father had a third nerve palsy. It was unnecessary to ask questions or make blood tests to diagnose familial syphilis, but this was purely incidental to her pneumonia. However, should her convalescence be protracted, the underlying hereditary syphilis might become pertinent and her improvement hastened by specific treatment.

The Wassermann test is unquestionably the most valuable laboratory test yet discovered, but to depend on it exclusively is to court disaster. There are three classes of physicians who attach undue importance to this test: first, the lazy physician who would rather

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some one else would make his diagnosis for him; second, the man incapable of taking a complete history and making a thorough examination, and lastly the type which Christian has so well described as the man who "with but a slight glance at his patient, and no conception of the patient's symptoms, rushes to his laboratory tests with the idea that scientific accuracy increases by the square of the distance from the bedside and by the cube of the time spent in carrying out a test."

To the dictum, "When in doubt have a Wassermann test," we should add, "but first make an intensive study of the family."

ABSTRACT OF DISCUSSION

DR. W. W. GRAVES, St. Louis: I have on several occasions called attention to the importance of studying the whole family where one of its members gives even the slightest indication of syphilis. One of the cases reported by Dr. Stoll, where he suspected syphilis in the third generation, was brought to him because the patient had interstitial keratitis, and he studied every member of the family and found that the parents and grandparents in that family were syphilitic. Dr. Kaskell, in discussing Dr. Drysdale's paper, stated that a recent study had shown the Wassermann reaction in 40 per cent. of the wives of paretics and tabetics was positive, whereas only a small number of the progeny of these paretics and tabetics gave positive Wassermann reactions. In our study of syphilis we are inclined to rely too much on positive and negative Wassermann or luetin reactions, whereas we should study the family, not only as far as the history of that family is concerned in all its details, but we should. as well, make comparative clinical studies of families. If there is one thing that is particularly characteristic of the progeny of syphilitic parents it is the deviation to be found in such progeny when compared with the parents. I grant that it is common to find children of syphilitic parents who compare favorably with other children of the community, but the only standard we have for the individual is how he compares with his ascendant, and those who are most accessible are the parents, and then a comparison of each child with the other. The characteristic thing of children born of syphilitic parents is that they do not compare favorably with their parents, particularly in their physical endowments and in their resistance to disease. We know how rare are Hutchinson teeth, how infrequent is interstitial keratitis, how rare is deafness without otitis, and how infrequent is the

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Wassermann reaction, but if we study every member of the family in a comparative clinical way we cannot fail to find gross deviations which demand explanation. The most pernicious effects of syphilis are on the parental germ plasm, from which the individual develops, hence the deviations, hence the vulnerability to disease of syphilitic progeny.

DR. A. L. SKOOG, Kansas City: I recently studied an interesting case, a girl of 18, who five months ago developed a hemichorea involving the right leg, arm and face. It was progressive. Outside of that there were practically no clinical findings; the reflexes were but very little different on the right side as compared with the left. A Wassermann was made on the blood which resulted in a 4 plus positive. The spinal fluid likewise showed a 4 plus positive. There was no lymphocytosis. The globulin content was increased. This girl's father and mother died when young, the patient being the only child. I could not find positive cause of death in either parent, but suspected vascular syphilis. Two sisters on the maternal side are living in the same city and are now being treated for late syphilitic troubles after certain positive diagnosis had been made.

DR. WILLIAM RAVINE, Cincinnati: I would like to ask Dr. Graves if the scaphoid scapula can still be looked on as one of the stigmata of hereditary syphilis, or if there has been reason to change the view concerning this finding. In the work of examining juvenile delinquents I always look and note the existence or absence of the scaphoid scapula, and very often we find nothing else of value.

DR. W. W. GRAVES, St. Louis: I have never stated that the scaphoid scapula should be considered a sign, in itself, of congenital syphilis in the individual or of syphilis in his parents. There is but one way to determine its significance, and that is to study every member of the same generation for the scaphoid scapula and what I have called its correlations, plus a similar study of the parents and other ascendants. The scaphoid scapula is of great value as a clew, but as evidence of congenital syphilis it should never be used alone. Since finding the scaphoid scapula eight years ago, and later indicating its clinical significance, I have feared that the profession might use the scaphoid scapula as a "pathognomic" sign of congenital syphilis, whereas nothing could be further from the truth.

DR. HENRY FARNUM STOLL, Hartford, Conn: I want to add my testimony to what Dr. Graves has just said in regard to the scaphoid scapulae. As examining physician of an outdoor school of a hundred pupils, in the course of a year I see a good many children and for over a year I have been on the lookout for scaphoid scapulae. I have been fortunate in having a social worker who has made intensive studies of the families for me and I agree with Dr. Graves that when

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it is possible to make a complete study of the parents and grandparents, one can nearly always find symptoms or signs strongly suggestive of syphilis. Occasionally it is the only stigmata. Fournier, who years ago said that paresis and tabes were due to syphilis, also stated that the heredo-syphilitics of the weakling group are especially prone to tuberculosis. When they develop this disease it is especially apt to be fatal. A Colorado physician has recently made a special study of a series of cases of tuberculosis that were doing badly and found that a high percentage showed various dystrophies. He did not deduce, however, as I think he should have, that these dystrophies were probably due to hereditary syphilis. We should commence to teach the public that syphilis is exceedingly common and that it is liable to occur in any family. We should teach them to look on syphilis as a misfortune, not as a disgrace, for there are more innocent than guilty sufferers. Until we promulgate this idea we will fall short of solving the problem of syphilis.

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EARLY DIAGNOSIS OF TABES DORSALIS*

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Tabes dorsalis, as its name would imply, is a disease characterized by a degeneration of the posterior columns of the spinal cord. Spiller,[1] however, as far back as 1897, defined tabes in reality as a cerebrospinal disease, and indeed, certain symptoms referable to the cranial nerves may so dominate the clinical picture that the cord symptoms may be insignificant or even pass unnoticed. Prominent among these symptoms are those referable to the eye, and especially primary optic atrophy.

Recently, E. Frey,[2] from Schaffer's clinic, has studied 286 cases of incipient tabes, classifying these as oculo-pupillary, dysuric, lancinating and mixed forms, and studying sixty of them also from the standpoint of prognosis.

The session[3] of the Society of Neurology in Paris, Dec. 30, 1911, was devoted to a discussion of the symptomatic limitation of this affection ("Délimitation du tabes"), and the prominence of the men who took part in this discussion makes it a worthy contribution to the recent literature of this subject. J. Nageotte maintained that tabes could be defined only by terms of pathologic anatomy and by the etiology, reiterating his radicular nerve theory. Nageotte's conception was accepted by such clinicians as Babinski and André

* From the Neurological Clinic, Leland Stanford Junior University School of Medicine.

1. Spiller, W. G.: The Pathology of Tabes Dorsalis, Int. Med. Mag., 1897, v, No. 5.
2. Frey, E.: Ueber klinische Formen, Symptomatologie und Verlauf der Tabes auf Grund von 850 Fällen, Ztschr. f. d. ges. Neurol. u. Psychiat., xvi, 1.
3. Rev. neurol., 1911, No. 24.

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Thomas, the latter, however, not regarding the radicular lesion as pathognomonic. The question of monosymptomatic tabes was raised; and although many of those taking part in the discussion held that an isolated symptom, characteristic of tabes in a patient with a history of syphilis and presenting a typical evolution, might make a diagnosis of tabes very probable, yet an absolute diagnosis was not justifiable. (Erb[4] in a recent communication elsewhere also expresses a similar opinion). Henri Dufour reported a necropsy case in which the only symptom during life was an Argyll Robertson pupil and in which the cord showed the characteristic lesions of tabes.

In answer to the question, how early one may venture to diagnose tabes, the quotation from John Babinski may not be out of order:

> A patient presents the sign of the Argyll Robertson pupil; furthermore, he is subject to lancinating pains showing the characteristics of those which occur in a plain case of tabes, and finally, notwithstanding the subjective pain, intensive faradization of the calf muscles is painless; in such a case, even in the absence of any other symptom, a diagnosis of tabes is almost sure. I can almost say as much of a patient in whom all the reflexes, including the pupillary reflex, are normal, and who presents together with the sensibility disturbance just noted a characteristic lymphocytosis of the cerebrospinal fluid. Certainly the diagnosis is not of a mathematical certainty, but of a great probability.

This statement, coming from a clinician of the prominence of Babinski, is noteworthy, and demands attention and further analysis. It is a long step from this picture to the picture presented by the classical tabetic: difficulty in walking, Romberg, ataxia, absent patellar reflexes, fixed pupils, advanced sensory disturbances and often typical complications. Syphilis is now regarded as the cause of tabes. Even Erb,[4] who has always recognized the important part which syphilis has played in this affection, has withdrawn from his former reserved position, and now states that syphilis is the sole cause outside of very exceptional

4. Erb: Tabes Deutsch. Ztschr. f. Nervenh., 1913, xlvii, 74.

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and doubtful cases. How syphilis acts on the spinal cord has given rise to many theories, the toxic theory, and the meningeal theories of Obersteiner and Redlich, and of Nageotte occupying the foreground of attention. A considerable support to the toxic theory has been given by the recent experiments of Spielmeyer.[5]

In order to determine to what extent chronic leptomeningitis of the spinal cord exists in cases of tabes which have been examined at necropsy, I have collected fourteen cases, examined microscopically, from the records of the Pathological Department of Cooper Medical College and Stanford Medical School between the years 1898 and 1916. Thickening or cellular infiltration of the spinal meninges most marked in the posterior portion of the cord was present in all of these cases. Recently Spiller,[6] in discussing the pathology of tabetic ocular palsy, has stated that in his experience the most characteristic lesion in cerebrospinal syphilis is round cell infiltration of the pia, and that he believes that there is no acute manifestation of syphilis of the cerebrospinal axis without it. This author examined sections from eleven cases diagnosed clinically as tabes, and found in all lymphocytic infiltration of the pia, this being marked in nine cases. He quotes Bresowsky, who found this picture constant in forty cases of tabes. Schröder[7] found in all of five cases studied a pial inflammation of inflammatory nature.

I may mention here the studies of Leri[8] on optic atrophy in tabes, in which this author believes that the pathologic process is an interstitial neuritis of the optic nerve due to syphilitic meningitis and syphilitic vascular disease, and the contribution of Vincent,[9] who

5. Spielmeyer, W.: Experimentelle Tabes bei Hunden, München. med. Wchnschr., 1906, No. 48, p. 2338.

6. Spiller, W. G.: Pathology of Tabetic Ocular Palsy, Jour. Nerv. and Ment. Dis., 1915, xlii, 15.

7. Schröder, P.: Ein Beitrag zur Histopathologie der Tabes dorsalis, Centralbl. f. Nervenh. u. Psychiat., Neue Folge, 1906, xvii, 585.

8. Leri, André: Etude du nerf optique dans l'amaurose tabétique, Nouv. iconog. de la Salpêtrière, 1904, No. 17, p. 356.

9. Vincent, V.: Des meningites chroniques syphilitiques, Thèse de Paris, Paris, G. Steinheil, 1910.

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studied the degenerations of the third, ninth, tenth, eleventh and twelfth pairs of cranial nerves in tabes and found, in the sense of Nageotte, a transverse radicular neuritis at the place where these nerves are included in the dural and arachnoid envelopes.

I am inclined to favor the opinion that the cause of the tabetic degeneration is a radiculitis subsequent to primary syphilitic meningitis, believing that this meningitis is sufficient to explain the posterior root degeneration. Whether this meningitis affects posterior roots in the locality favored by Obersteiner and Redlich,[10] or by Nageotte,[11] although interesting from the histopathologic point of view, is of secondary practical importance: the important point to my mind is whether or not the tabetic process starts as an active inflammatory one in the meninges. The importance of a clear understanding of this question is primary, not alone on account of the great diagnostic importance of an inflammatory cerebrospinal fluid, but also from the standpoint of local intraspinal injections by specific remedies.

SYMPTOMATOLOGY

As a basis for a clinical study of the early tabes, I have selected and tabulated thirty-five cases from a total of 150 studied, 119 of these cases being similarly tabulated. Tabulated cases cannot be recorded as completely as separate case reports, but this table presents in graphic form the most prominent features of early tabes. The following discussion is limited to certain of these features; for the general symptomatology, further reference may be made to the table. This list of early tabes is manifestly too small to be of value as a statistical study as regards sex, age, occupation, nationality and the incidence of syphilis as an etiologic factor, but it may be mentioned in passing

10. Obersteiner and Redlich: Ueber Wesen der Pathogenese der tabischen Hinterstrangs Degeneration, Obersteiner Arbeiten, 1894; abstr., Neurol. Centralbl., 1894, No. 12, p. 454.
11. Nageotte, J., and Riche, A.: Tabes dorsalis, Manuel d'histologie pathologique, Cornil et Ranvier, Ed. 3, 1907.

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that in this list of thirty-six cases, sixteen patients denied syphilitic infection. Of this number the Wassermann was negative in the blood or cerebrospinal fluid in only six cases. In this series of six cases, only two (Cases 13 and 36) showed an absence of pupillary signs—Argyll Robertson pupils, anisocoria, and pupils of irregular contour, which we have come to regard as characteristic in syphilis.

Sensibility.—Among the complaints, some kind of pain was the most frequent. Under the headings of lancinating pains and pains described as rheumatism, we group the greatest number of these symptoms; our records show that only three cases — these all being cases of pronounced optic atrophy — gave no history of pain or paresthesias. Headache was a common symptom, being present in ten cases. Passing from the subjective complaint of pain to the objective disturbances of sensibility, we have grouped these disturbances under the headings deep and superficial sensibility. I shall not detail the results of these examinations, nor are they tabulated, reserving this study for a future communication. The frequency of objective sensibility disturbance is parallel with the severity of the disease, and these disturbances are probably a constant symptom in tabes, negative results being due to the inefficiency of our method of examination. There appears to be no constant relationship between the presence of a positive Romberg symptom and a disturbance of the deep sensibility: notion of position, passive motion, localization, deep pressure sense, discrimination between two points and the vibration test. The loss of deep pressure pain brought out in Abadie's sign and Biernacki's sign were frequently present. Occasionally in testing for Abadie's sign hyperesthesia was present.

Superficial Sensibility: The radicular disturbance of the cutaneous sensibility noted by Leyden, Marie, Dejerine, Redlich, Massary and Brissaud has been

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particularly studied by Marinesco,[12] and by Patrick[13] in this country with reference to tactile anesthesia about the trunk. We have also noted the sensory involvement about the trunk and especially the increased sensitiveness to cold, and the girdle sensation. Ulnar paresthesias, diminished sensibility about the genitals and the anus has also been noted in our cases. In the lower extremities, however, the radicular disturbance of the sensibility has been in our experience less evident; in this locality the charts show a picture commonly seen in polyneuritis, or that described as a segmental distribution involving the feet and shading from the distal to the proximal end of the member. Marinesco[12] previously called attention to this point. The more frequent involvement of the superficial sensibility than the deep would be the opposite of what was to be expected in an involvement of the posterior columns of the cord only, which are not tracts for pain. The symptoms of pure posterior column involvement which, according to Dejerine,[14] is seen best in combined sclerosis, is not the picture here. We believe that the frequency and the constancy of pain in tabes is one of the most convincing proofs of the radicular or meningeal cause of the disease.

Reflexes.—I wish to call attention particularly to the state of the tendon reflexes in the lower extremities, the tendon reflexes in the upper extremities being generally conserved in our series contrary to the opinion held by Frankel. Preservation of the patellar tendon reflex and the Achilles tendon reflex is compatible with a diagnosis of tabes: in thirteen of our cases such a condition was present. The loss of the patellar tendon reflex or Westphal sign is properly considered

12. Marinesco, G.: De la topographie des troubles sensitives dans le tabes; ses rapports avec les sensations des tabétiques, la sem. méd., 1897, No. 47, p. 369.
13. Patrick, H. T.: Anesthesia of the Trunk in Locomotor Ataxia, New York Med. Jour., Feb. 6, 1897, p. 173.
14. Dejerine, J.: Le syndrome des fibres radiculaire longues des cordons postérieurs, Presse méd., 1913, No. 104, p. 1053.

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an important and characteristic symptom of tabes. I wish particularly to discuss the state of the Achilles tendon reflex in these early cases. Babinski[15] (1901) deserves the credit of calling attention to the early loss of this reflex, which he claimed generally disappeared before the patellar tendon reflex. During the period 1898-1901 this author observed forty cases of this nature, as opposed to five cases in which the patellar tendon reflex was absent and the Achilles tendon reflex present. Prior to Babinski's publication, Goldflam[16] noted the absence of this reflex in cases of tabes, and Ziehen[17] discussed its diagnostic signification in various psychoses and psychoneuroses. Following Babinski's first communication in 1898, C. K. Mills[18] found this reflex present in only three cases of tabes out of twenty-eight cases, in all of which the patellar tendon reflex was present. Van Gehuchten,[19] Biro[20] and Tumpowski[21] arrived at similar results. Kollartis[22] found eleven cases in 100 in which the Achilles tendon reflex was lost and the patellar tendon reflex conserved; and in only one case was the opposite the case: Achilles tendon reflex present, patellar tendon reflex absent (unilaterally). Sarbo,[23] in ninety-two cases, found 91 per cent. missing Achilles tendon reflexes, and in 190 cases 80.4 per cent. missing patellar tendon reflexes. In seven cases the Achilles tendon reflex was lost, the patellar tendon

15. Babinski, J.: Sur le tendon d'achille dans le tabes, Société médicale des hôpitaux, seance du 21. Oct., 1898. Société de neurologie de Paris, Séance du 2, Mai, 1901; abstr., Rev. neurol., 1901, ix, 482.

16. Goldflam, S.: Ueber die Ungleichheit der Knie Phänomene bei Tabes dorsalis, Neurol. Centralbl., 1888, pp. 529, 556.

17. Ziehen, T.: Zur diagnostischen Bedeutung des Achilles-Sehnenphänomens, Deutsch. med. Wchnschr., 1894, No. 34, p. 670.

18. Mills, C. K.: Some Points of Special Interest in the Study of the Deep Reflexes in the Lower Extremities, Jour. Nerv. and Ment. Dis., 1899, xxvi, No. 3.

19. Van Gehuchten, quoted by Babinski: Footnote 15.

20. Biro, M.: Ueber Störungen des Achillessehnen Reflexes bei Tabes und Ischias, Deutsch. Ztschr. f. Nervenh., xix, 188.

21. Tumpowski, A.: Beitrag zur Aetiologie und Symptomatologie der Tabes dorsalis, Deutsch. Ztschr. f. Nervenh., 1897, p. 467.

22. Kollartis: Das Verhalten einiger Reflexe bei Gesunden und bei Tabes, Deutsch. Ztschr. f. Nervenh., 1902, No. 23, p. 89.

23. Sarbo, A. von: Klinische und statische Daten zur Symptomatologie der Tabes, Deutsch. Ztschr. f. Nervenh., 1902, No. 23.

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reflex conserved. Förster[24] and Strassburger[25] noted the absence of this reflex in tabes, without, however, calling attention to its relative importance. Bregman[26] draws an analogy between the loss of this reflex and cutaneous sensibility disturbances in the area supplied by the fifth lumbar and first sacral roots. Oppenheim[27] sounds a warning note against attaching too great importance to the absence of this reflex in the diagnosis of tabes, because of its frequency in sciatica and related peripheral nerve conditions. Jelliffe and White[28] state that the Achilles tendon reflex is not infrequently lost before the patellar tendon reflex.

The technic of eliciting this reflex is important, the kneeling position with the foot hanging free and relaxed being necessary for a proper performance of the test (Babinski). In our series there were twenty cases in which one or both of the Achilles tendon reflexes were absent with conserved patellar tendon reflexes, and in seven of these, the Achilles tendon reflex was absent on one side only. A striking relationship in regard to the absence of this reflex was seen in ten cases of primary optic atrophy, the extremities of the cerebrospinal axis, as it were, being affected by the tabetic process. It would then appear that the loss of the tendon reflexes in the lower extremities in tabes takes the form of a regular progression from below upward in the majority of cases, and in this regard Cases 33 and 36 seem to show both Achilles tendon reflexes lost before the patellar tendon reflex, the latter reflex being lost first on one side only. Further, in reviewing the total number of cases (150) we have encountered only one instance in which the patellar ten-

24. Förster, O.: Zur Symptomatologie der Tabes dorsalis, Monatschr. f. Psychiat. u. Neurol., viii, 1.

25. Strassburger, J.: Ueber das Fehlen des achillessehnen Reflexes und seine diagnostische Bedeutung, Deutsch. Ztschr. f. Nervenh., xvii, 315.

26. Bregman, L.: Sehnenreflexe und Sensibilitätsstörung bei Tabes dorsalis, Neurol. Centralbl., 1905, No. 1, p. 2.

27. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, 1913, Berlin, Karger.

28. Jeliffe and White: Diseases of the Nervous System, 1915.

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don reflex was absent when both Achilles tendon reflexes were conserved, and in this one instance only one patellar tendon reflex was lost. From these data, then, we believe that as a rule the loss of the Achilles tendon reflex precedes the loss of the patellar tendon reflex, and is therefore an earlier and more important diagnostic sign. It is possible in the cervical type of tabes that a different order may ensue, but such an example has not yet come under our observation.

In our cases we note bladder disturbance in ten cases, a very important early sign of cord involvement; also gastric crises in ten cases. The frequency of general glandular enlargement including epitrochlear enlargement (eleven cases), and cardiovascular disease is called attention to because they are important symptoms of syphilis and not of tabes. In fifteen cases psychic or neurotic disturbances were noted.

Eye Symptoms.—The frequency of eye symptoms in tabes has long been recognized, and among these, primary optic atrophy is probably the most frequent. The remarkable statistics of von Grosz[29] show 88 per cent. of diseased papillae in 100 cases examined, and in only two out of the twelve normal backgrounds were present normal vision, normal fields and normal color perception. In thirteen of our tabulated cases primary optic atrophy was found, being quite advanced in a number of these cases. From the study of these we were able to pick out many of the early forms of tabes enumerated here. Argyll Robertson pupils and those showing definite sluggish reaction to light were present in twenty-four out of twenty-five cases; and anisocoria and pupils of irregular contour were found, respectively, in fifteen and thirteen cases which establishes these two conditions as of great importance as characteristic tabetic eye signs possibly antedating the Argyll Robertson pupil.

29. Von Grosz, quoted by Karl Schaffer: Handbuch der Neurologie, Lewandowsky, Spezielle Neurologie, 959.

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Ear Symptoms.—There were sixteen cases of diminished hearing, nine of these in association with optic atrophy. The absence of special examinations in all cases to determine the character of the deafness, whether a nerve deafness or of peripheral origin, is of course not conclusive; but recent observers, such as Knick and Zaloziecki[30] and Swift,[31] who called attention to syphilitic meningitis as the cause of diminution of hearing in certain cases of early syphilis, is important in suspecting a large percentage of our cases to be due to nerve deafness, consequent on radicular nerve disease. The fact that syphilitic disease of peripheral nerves is a very rare disease would support this view.

Trauma.—In eleven cases a history of trauma was obtained. The question of trauma as an exciting cause or a contributory cause in tabes in relation to disability has come up in industrial accident cases in California. An analysis of these eleven cases shows four cases (Cases 1, 10, 24 and 31) were injured before syphilitic infection took place. In two other cases (Cases 16 and 26) of failing eyesight and optic atrophy, trauma preceded the failing eyesight by so short a time that its rôle in the development of the optic atrophy was very questionable.

Wassermann Reaction and Cerebrospinal Fluid Analysis.—The Wassermann reaction was found positive in twenty cases in the blood and in twenty cases in the cerebrospinal fluid, and in one or both in twenty-seven cases. The globulin reaction was present in twenty-two cases out of the thirty-one done. The cell count was positive in twenty-seven out of thirty-two cases and was the most frequent positive cerebrospinal fluid reaction in our series. In five cases the cell count was not increased, three cells being regarded as the upper limit of normal per cubic millimeter. In a

30. Knick, H., and Zaloziecki, A.: Ueber Acusticuserkrankungen in Frühstadium der Lues, Berlin. klin. Wchnschr., 1912, xlix, Nos. 14, 15.
31. Ellis, A. W. M., and Swift, H. F.: Involvement of the Eighth Nerve in Syphilis of the Central Nervous System, THE JOURNAL A. M. A., May 1, 1915, p. 1471.

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recent paper[32] on the subject of cerebrospinal fluid analysis in which 116 fluids were examined, it was my opinion that six cells per cubic millimeter were the upper limit of normal, but we have come to regard this figure as too high. The lower figure corresponds with the results of Nageotte, who found an average of three cells per cubic millimeter in fifty-six normal fluids examined. I myself prefer the Nageotte[33] counting cell, and count at least six cubic millimeters of fluid, making two separate spreads and being careful to obtain a good admixture of fluid and to count the fluid a short time after it is withdrawn. It is exceedingly likely that variation in results is often due to a failure in technic. In three cases the cells and globulin were not increased, and in two cases all reactions of the cerebrospinal fluid, including the Wassermann test, were negative. Nonne[34] reports eleven such cases in 167 cases of tabes. In Case 13 we have a record of an entirely negative cerebrospinal fluid with definitely thickened membranes.

This brings us to the consideration of the importance and constancy of the cerebrospinal fluid reactions indicating inflammation: increased cell count and increased globulin content and probably also the Lange gold test. Since Widal, Sicard and Ravaut[35] found an increased cell content in cases of paresis and tabes, and Guillian and Parant[36] noted an increased globulin content, these tests have occupied first rank as important diagnostic aids in detecting syphilis of the central nervous system. In the Paris meeting noted above it was the opinion of Sicard that a lymphocytosis was

32. Schaller, W. F.: A Report on the Analysis of the Cerebrospinal Fluid, Jour. Nerv. and Ment. Dis., 1913, xl, No. 8.

33. Nageotte and Levy, Valensi: Numeration direct des élements cellulaires du liquid cephalorachidien, Compt. rend. Soc. de biol., 1907, ii, 603.

34. Nonne: Der heutige Standpunkt der Lehre von der Bedeutung der vier Reaktionen, Deutsch. Ztschr. f. Nervenh., 1911, xlii, 201.

35. Widal, Sicard and Ravaut: Cytologie du liquide cephalo-rachidien au cours de quelques processus méningés chroniques (paralysie générale et tabes), Bull. et mém. Soc. méd. d. hôp. de Paris, 1901, 18. janvier, p. 31.

36. Guillian and Parant: Sur la présence d'albumines coagulables par la chaleur dans les liquid cephalo-rachidien des paralytiques spinaux, Rev. neurol., April 30, 1903, p. 406.

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constant in tabes as a rule. Soques had found it absent in one or two times in tabes. André Leri considered the lymphocytosis as indicating a meningeal process in evolution, and reported on the examination of a spinal cord in which at different levels the histologic picture varied; in the cervical region thickened membranes and infiltration of leukocytes, whereas the dorsal cord showed definite thickened meninges without infiltration of leukocytes.

This brings us to the important point of correlating our findings in the cerebrospinal fluid with our conception of the histopathology of tabes dorsalis. We have seen in our list of necropsies that a chronic syphilitic leptomeningitis, most intense in the posterior aspect of the cord, is a constant finding. It would be only natural to assume that such an inflammation in the meninges would manifest itself in increase of cells and globulin in the cerebrospinal fluid. I believe that repeated lumbar puncture will show at one time or another increased cell count and albumin in a progressive tabes, and that in a negative outcome of such repeated tests the diagnosis of tabes should always be made with reserve. It may not be amiss to report a case[37] previously but erroneously reported as tabes, which presented certain characteristic symptoms of tabes: gastric crises, ataxia, sensory disturbances and absent Achilles tendon reflexes and patellar tendon reflexes. Repeated examinations of the cerebrospinal fluid were constantly negative as regards cell count and globulin, but finally a faintly positive Wassermann reaction in the cerebrospinal fluid was obtained. Syphilis was denied. This patient subsequently died of an intercurrent affection, and necropsy revealed posterior column degeneration on cut sections of the cord which led to the diagnosis of tabes dorsalis; but the meninges were not thickened or infiltrated. This feature was commented on, and this was considered a case worthy to report, but Weigert's stain for myelin degeneration showed the typical picture of a combined sclerosis. I

37. Schaller, W. F.: Salvarsan in Syphilitic Disease of the Nervous System, California State Jour. Med., October, 1912, Case 10 (Tabes).

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am not aware of any case of tabes being yet reported which has shown clinically a negative cerebrospinal fluid finding for cells or globulin, and in which a following postmortem examination has revealed normal spinal meninges by microscopic examination.

CONCLUSIONS

1. In tabes the syphilitic posterior leptomeningitis of the spinal cord (and probably also of the radicular nerve) is in etiologic relationship to the degeneration of the posterior columns of the cord. Every case of syphilitic posterior leptomeningitis and consequent multiple symmetrical radiculitis is a case of potential tabes.

2. This meningeal reaction is evidenced in the cerebrospinal fluid by an increased cell count and increased globulin content; and probably also by a positive Wassermann reaction indicating that this reaction is syphilitic in nature. During certain stages in the evolution of tabes, probably of repair, these reactions may be negative. It is therefore necessary to make repeated lumbar puncture in doubtful cases.

3. Syphilitic radiculitis has its characteristic disturbance of the sensibility: Subjective disturbances are pain and paresthesias; objective disturbances are deep and superficial sensibility impairment, the latter following radicular distribution. In the lower extremities, this radicular involvement cannot always be demonstrated, the sensory cutaneous involvement often presenting a peripheral or segmental type.

4. The Achilles tendon reflexes are lost as a rule before the patellar tendon reflexes: this phenomenon was observed in ten cases of primary optic atrophy from a total of thirteen cases.

5. Anisocoria and pupils of definitely irregular contour are found frequently in early tabes. These pupillary signs, even in the absence of the Argyll Robertson pupil, are highly characteristic of early tabes, provided there is no local assignable cause.

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6. The symptom of diminished hearing is frequently encountered in early tabes.

7. Cardiovascular disease, especially aortic disease, and general glandular enlargement are frequently found in tabes, thus giving evidence of syphilitic processes elsewhere in the body.

8. In a patient with a history or other evidence of syphilis presenting characteristic sensibility disturbances of the radicular type with a tendency to symmetry, one should suspect a potential or early tabes. If, associated with the foregoing, we have a positive reaction in the cerebrospinal fluid indicating a chronic syphilitic meningitis, together with such pupillary phenomena as anisocoria, pupillary irregularity or sluggish reaction to light, the diagnosis of early tabes is most probable. Added to the foregoing symptoms, the loss of the Achilles tendon reflex establishes the diagnosis of early tabes even in the absence of those signs which we usually associate with tabes: Romberg, marked sensibility loss, absent patellar reflexes and Argyll Robertson pupils.

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SHALL WE TREAT THE PARETIC?

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The first clear presentation of the clinical relationship of the mental and physical symptoms in paresis was made by Bayle in 1822, although the earliest description of this disease was given toward the close of the eighteenth century by Chiarugi and Haslam.[1] Almost forty years ago, Fournier startled and antagonized the medical world by suggesting that syphilis in the great majority of cases was the cause of locomotor ataxia. This has been a subject of bitter discussion for more than a third of a century; in fact it was Metchnikoff's infection of apes with syphilis in 1903 that robbed syphilis of its mystery, and gave it a definite standing among infectious diseases. This was the first definite step in the elucidation of our present knowledge of the pathology and symptomatology of nervous syphilis. Then came notable years. Schaudinn in 1905 discovered the *Spirochaeta pallida*. In 1906, Wassermann announced his reaction, which proved the presence of syphilis in the varied manifestations of syphilis. The researches of Noguchi, Moore, Marie and Levaditi, who have found spirochetes in the lesions of tabes and paresis, demonstrated that in these affections, we are dealing with actual syphilis: a late manifestation of the infection. It has been recently suggested that we discard the terms "paresis" and "tabes," and speak only of cerebrospinal syphilis, par-

1. Chiarugi and Haslam, quoted by Kraeplin: General Paresis, Nervous and Mental Diseases, Monograph Series, No. 14, 1913.

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etic or tabetic in type. The yearly economic loss to New York State on account of insanity is about $35,000,000. Syphilis was the principal cause of insanity in 19.8 per cent. of the male cases and in 7.2 per cent. of the female cases admitted to New York State hospitals in 1913. "Of the killing diseases," says Sir William Osler, "syphilis comes third or fourth." Church states that it is a conservative estimate that one man in every five is syphilitic. In the New York State hospitals, paresis constitutes one eighth of all the admission. Out of 4,000 syphilized men examined, twenty-two years after the last of them was infected, Mattauschek and Pilcz[2] found that 6.27 per cent. had developed paresis or tabes or both of these diseases. It has been estimated that from 9 to 25 per cent. of those infected develop nervous symptoms. At the San Francisco session of this Association, we placed great emphasis on the importance of the examination of the spinal fluids in early syphilis. Hoffman, Nicols and Steiner have observed that in 15 per cent. of the cases in primary and secondary syphilis, the spinal fluids are infectious, and infection by their inoculation can be obtained before changes can be observed in the Wassermann and other reactions.[3] After a syphilitic infection, it would appear that there is invariably an involvement of the central nervous system, even though it be of slight duration and unattended by neural change. That a variation in the spirochete occurs is shown by laboratory experiments. Wile[4] observed that spirochetes injected into rabbits from the living paretic brain were of a virulent type, and that the incubation was shorter than with other strains. Investigators in the laboratory of the Army Medical School in Washington have bred several varieties of spirochetes.

2. Mattauschek and Pilcz: Ueber die weiteren Schicksale 4,134 katamnestische verfolgte Falle luetische Infektion, Med. Klin., 1913, ix, 1544.

3. Church, Archibald: Some Modern Views of Syphilis of the Nervous System, THE JOURNAL A. M. A., May 20, 1916, p. 1596.

4. Wile U. J.: Experimental Syphilis in the Rabbit Produced by the Brain Substance of the Living Paretic, Jour. Exper. Med., 1916, xxiii, 199.

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"They can breed a variety which will produce a hard chancre, or a variety which will produce a chancre lacking the old hunterian characteristics of a saucer-like indurated base. They can produce a spirochete which will give a mucocutaneous efflorescence, or a spirochete which will be poor in cutaneous manifestations, with a tendency to confine its influence to the nervous system" (Church).

During the past decade, our conception of syphilis and its therapy has been absolutely revolutionized. The latter was based largely on hypothesis and empiricism. Twenty-seven years ago, we were taught that the essential and primitive anomaly in the paretic's brain was "a vasomotor disturbance." [5] Trephining was therefore suggested by Batty Tuke of Edinburgh, to overcome cerebral congestion and we (Dr. Riggs) can well recall the enthusiasm with which he discussed the potentialities of the new procedure. This was the first step in the subdural medication of today. In 1901, Dr. Ford Robertson,[6] suggested that paresis was a disease due directly to the toxins of bacteria, the point of attack being the alimentary tract. In 1903, he first announced his theory of *Bacillus paralyticans* as a cause of dementia paralytica.[7] In 1905, Robertson and McRae[8] reported the presence of the diphtheroid bacilli in the genito-urinary tract in general paralysis and tabes dorsalis. The same authors,[9] in 1907, discussed the treatment of paresis and tabes by vaccines and antiserum. While they first tried vaccines, they placed their reliance on the antiserum. The latter was obtained from immunized sheep which had been inoculated with dead cultures of the *Bacillus paralyticans-longus*, isolated from the brain of a general paralytic. After

5. Spitzka, E. C.: Manual of Insanity, 1889.
6. Robertson, Ford: Brit. Med. Jour., 1901.
7. Robertson, F. W.: Histologic Evidences of the Presence of an Organism Resembling the Klebs-Loeffler Bacillus in Cases of General Paralysis of the Insane, Rev. Neurol. and Psychiat., 1903.
8. Robertson, F. W., and McRae, G. D.: Observations on the Treatment of General Paresis and Tabes Dorsalis by Vaccines and Anti-Sera, Rev. Neurol. and Psychiat., 1907, v, 673.
9. Robertson, F. W., and McRae, G. D.: Rev. Neurol. and Psychiat., 1907.

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two months' treatment of the sheep, the serum was fit for use. It was administered by mouth, nose or hypodermic. Of twelve cases of paresis treated by them, they report that "all have shown remarkable signs of improvement." In tabes the ataxia was much benefited.

The favorable influence of an intercurrent acute febrile disease in certain cases of general paralysis has long been a matter of common observation. Since it would be neither judicious nor wise to infect the patients with a serious specific fever, other means for inducing a harmless temporary febrile reaction have been suggested. The tuberculin treatment of von Wagner[10] and the sodium nucleinate injections of Donath are the two classical methods of inducing artificial pyrexia in this disease. In 1909, von Wagner announced his treatment of general paralysis by injections of tuberculin (Koch) combined with mercurials and iodids. His pupil, Pilcz, in 1910-11, reported eighty-six cases of clinically demonstrated paresis which were treated by this method. Small doses, from 0.0005 to 0.01 c.c. of tuberculin were injected every second day, the dose being gradually increased until 0.3 or 0.5 or even 1 c.c. was administered. In 39.44 per cent. there was no perceptible or only a slight improvement, while in 60.32 per cent. there was a distinct clinical betterment observed. Remissions were noted in 26 per cent. of the cases. In 1903, Donath[11] reported six cases of paresis and of cerebral syphilis treated by salt infusion, given every third or fifth day. The infusion was administered for the purpose of freeing the system of toxins formed by the degenerating nervous substances. All these patients were improved by the treatment, and he believed the subcutaneous injections of salt solution of particular value

10. Von Wagner: Ueber die Behandlung der Progressiven Paralyse, Wien. med. Wchnschr., 1909, No. 27; International Medical Congress, Budapest.

11. Donath, Julius: Die Behandlung der progressiven Paralyse sowie toxischer und infektiöser Psychosen mit Salz Infusion, Deutsch. med. Wchnschr., 1903, xxix, 110.

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in early paresis. In 1910, he suggested the use of sodium nucleinate in place of the salt infusion.[12] He injected 1 gm. of this salt (a 2 per cent. solution in normal saline) once a week. There is a rise of temperature, which attains its maximum in from four to ten hours and returns to the normal in from one to two days; a transient leukocytosis numbering from 10,000 to 25,000 per cubic millimeter occurs, which disappears generally by the fifth day. He reported the treatment of fifteen patients with paresis; 60 per cent. showed improvement in tremor and memory and a lessening of acute mental symptoms; three were able to return to work; six were improved; in five there was no change; one died of apoplexy. Mercury was also used.

Last fall, we (Dr. Riggs) wrote twenty-six of our leading neurologists and alienists in regard to their experience with the pyrexia treatment in paresis. None had used tuberculin; two, Dr. Dana and Dr. Barrett, had tried the sodium nucleinate injections. The former stated that he had used the nucleates of soda in several cases of paresis. He had not seen it do any good and had not heard of any good results. The latter wrote: "We did a little work with the nuclein treatment of Donath, but our work was not carried very far. I felt that with such methods as these we could not get at the real root of conditions present in paresis." In Dr. Church's reply, he said: "Many years ago (twenty-five years) some such cases were treated with inoculations of modified erysipelas germs, or what amounted to that, in the hospital at Elgin, only, however, with unfortunate results." Regarding this method, Dr. Purves Stewart says: "I have made a number of observations with a moderate amount of encouragement." Dr. C. W. Burr questions the beneficial influence of an acute febrile attack in these patients. "I have seen a few patients, who, during a febrile attack, seemed to tem-

12. Donath, Julius: Weitere Ergebnisse der Behandlung der progressiven Paralyse mit Natrium nucleinum, Berlin klin. Wchnschr., Dec. 19, 1910.

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porarily improve. My impression is that the cause of the fever was of no importance. I have never seen any permanent good result. I have seen many other patients with paresis in whom fever had no influence at all."

In 1905, Dr. Myer[18] said, "The important point about general paresis seems to be finding out what makes general paresis a progressive disease." Modern research answered that question. The important problem now is how to destroy the spirochete. The potentiality of our therapeutic resources has been greatly enhanced by Ehrlich's discovery of salvarsan in 1909. It is the consensus of opinion that all the varied manifestations of syphilis can best be treated by the combined use of mercury and salvarsan. Since these patients first consult the general practitioner, on him rests a grave responsibility, for only by an early recognition of the initial lesion and a radical use of modern methods of therapy can a cure be obtained, or late syphilis (paresis or tabes) be prevented. After invasion of the nervous system, one of two modes of procedure may be advised: intrathecal or intensive medication. To Marinesco, in 1911, belongs the credit of the initiation of the former. Swift and Ellis used salvarsanized serum intraspinally in the therapy of tabes dorsalis in 1912, while Robertson of Edinburgh employed it in paresis in 1913. In 1914, Ravaut injected neosalvarsan directly into the spinal fluid. Byrnes during this year used intraspinal injections of mercurialized serum. In 1915, Hammond and Sharpe treated three paretic patients by direct injection into the lateral ventricles of salvarsanized serum and neosalvarsan added to the serum.

This multiplicity of procedure is not chaotic, as it would at first appear, it is an expression of the scientific purpose to reach the hitherto inaccessible spirochetes, to bring the spirocheticidal solution into con-

13. Myer Dana, Charles L.: The Curability of Early Paresis, THE JOURNAL A. M. A., May 6, 1905.

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tact with the infecting organism; it is simply science groping, if somewhat blindly, for a definite result. "The end in view in this treatment of nervous syphilis," say Swift, "is threefold, i. e., cure, amelioration or prolongation of life." Have modern methods, as employed in paresis, been of value in any of these respects? Certainly not in cure in fully developed types of the disease. Do they arrest or favorably influence paresis or prolong life? Many believe that as good results were obtained from the routine treatment of the presalvarsan period. Others regard modern methods as most disappointing. Evans and Thorne[14] state that as much improvement may be seen in patients who have received no special treatment as they observed in their cases following the combined use of the intraspinal and intracranial methods. Such, however, has not been our observation or experience.

We believe that, by intraspinal medication, remissions are greatly increased in frequency; that in early paresis, marked improvement may occur, with a possible arrest of symptoms. Paine[15] has observed 1,279 untreated cases in which there occurred ninety-five remissions. Statistics vary greatly as to their occurrence, varying anywhere from 4 to 20 per cent. Cotton has observed as the result of treatment 35.5 per cent. of remissions, while in the untreated cases, the number of spontaneous remissions was about 4 per cent. Ogilvie[16] has reported twenty-six remissions, averaging over a year each, out of thirty-five cases. The minimum number of injections required to induce a remission was six; the maximum, fourteen. "The establishment of a remission," says this writer, "is something gained, but it is merely one step in the right direction." The favorable results of this treatment, according to Amsden, can best be expressed in terms of remission. "The value of remissions in general

14. Evans, B. D., and Thorne, F. H.: The Treatment of Paresis, Am. Med.-Psychol. Assn., 1915, xxii.
15. Paine, L. H.: Boston Med. and Surg. Jour., 1913, clxviii, 648.
16. Ogilvie, H. S.: New York Neurological Society, Jour. Nerv. and Ment. Dis., March, 1916.

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paresis in any line of treatment is difficult to estimate," say Barrett. "We have, however, gained the impression from a considerable amount of work, that remissions are more complete and patients are able to remain out of the institutions longer when treated intraspinally than by any other mode we have used."

Regarding the arrest of the paretic process, Marinesco and Minea[17] say:

> Modern therapeutics cannot accept the dogma of the incurability of general paresis, not even of its steady progress; there are cases known where the disease is stationary; such are rare. There are many cases in which repeated remissions occur. The stationary state, as well as the repeated remissions, prove not only that the lesions can stop for some time, but also that there must be in the brain of general paralytics alterations which can be replaced.

These writers suggest that remissions are due to the formation of antibodies as in other infectious diseases.

We have had the opportunity of observing seventy-one cases of paresis during the past four years, of which twenty-one have been treated by us. Of the fifty untreated cases, or those treated intravenously or with mercury, one only has had a remission. The brief citation of four cases will be sufficient.

Case 1.—Man. The grandiose type, with characteristic spinal blood and fluid findings of paresis. A remission of four months' duration occurred after the sixth treatment. A relapse occurred and after further treatment there was a second remission of five months' duration, which still persists. He is steadily improving and is practically normal, aside from occasional periods of mild elation. This patient has had forty-one intraspinal injections (thirty-four salvarsanized serum; seven mercurialized serum) with no untoward symptoms. The spinal fluid gives a positive Wassermann in 0.8 c.c.; there is a trace of globulin; no lymphocytosis; blood Wassermann negative; no change in the colloidal gold reaction.

Case 2.—Man, aged 44; family and personal history negative; aside from alcoholism. Acute grandiose type; patient showed mental unballance a year before coming to us. Biologic findings typically those of paresis; remission took place

17. Marinesco and Minea: L'emploi des injections de serum salvarsanisé "in vitro" et "in vivo" sous l'arachnoid spinale et cérébrale dane le tabes et la paralyses générale, Rev. Neurol., 1914, xxvii, 337.

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after the seventh injection, which lasted for eight months. He was able to resume the oversight of his business affairs. He then became homicidal and suicidal and was sent to the state hospital. Aside from the suicidal propensity, his mental condition seems good. He received twenty-one injections of salvarsanized serum (Swift and Ellis); blood Wassermann negative; spinal fluid negative in 0.8 c.c.; positive in 1 c.c.; no globulin; no lymphocytosis; colloidal gold curve uninfluenced.

Case 3.—Man, aged 48; single; gonorrhea in 1900; denied syphilis; when seen by us he was acutely excited; it required four men to bring him to the hospital. Since January, 1915, patient has been indifferent about his work and restless. He became elated in March, 1915. There were Argyll Robertson pupils and increased knee jerks. Serobiologic findings those of paresis. After the sixth injection of salvarsanized serum, he was able to return home and help on the farm. At present writing, aside from slight nervousness, he is apparently normal. He received in all twenty-six intraspinal treatments (Swift and Ellis). After a time, we gave him weekly, in addition, 2 grains of mercuric salicylate intramuscularly. Blood serum is negative; spinal fluid positive in 0.8 c.c.; no lymphocytosis; no globulin; gold curve unchanged.

Case 4.—Man, aged 38, contracted syphilis in 1902; consulted us, March, 1916; was talkative, disoriented, very restless, expectorated constantly and made grimaces; Argyll Robertson pupil, increased knee jerks and Babinski. Biologic findings those of paresis; was given mercurialized serum intraspinally with intramuscular injections of mercury. He grew gradually worse and died, April, 1916. No biologic reduction. Evidently this was a case of so-called "galloping paresis."

Of the twenty-one patients treated with salvarsanized serum intraspinally (Swift and Ellis) with and without mercury intramuscularly, ten showed a decided improvement. Of these, five are at present attending to their business affairs; one had a remission of eight months; another one of four months; one has had two remissions of four and five months' duration, respectively; one is at home. In this patient, there is a slight improvement with an occasional epileptic attack. Still another is home and is able practically to attend to his business. Three died, not following an injecton, but some time after discontinuing treatment. The eight remaining are still living and have mani-

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fested some improvement physically, but no change in the mental condition. In all but two of these cases which were sufficiently treated, the Wassermann in the blood became negative, but none gave a negative Wassermann in the spinal fluid; lymphocytosis disappeared; globulin a trace. We have been unfortunate that in none of our cases have we been able to render the spinal fluid negative. According to Ogilvie, it is practically impossible to get a negative Wassermann in true parenchymatous disease of the brain. When obtained, it indicates that the specific process is confined largely to the interstices of the cerebral tissues.

"At this hospital, we have treated, in all," says Dr. Blumer,[18] "twenty-five cases, twenty paretics and five tabetics (December, 1915). Of the paretics, five are now dead, four having shown no improvement; one died of a convulsion after having shown considerable improvement. In five other cases, the course of the disease continues unabated. The remaining ten have all shown great clinical improvement and have been able to return to their homes and business. Of these, three men have had no recurrence during nearly two years' absence from the hospital. The laboratory results have been rather discouraging. Only four patients have obtained a persistently negative Wassermann. Most of the others show no change, while in some the Wassermann has appeared and disappeared irregularly. Three show disappearance of the gold chlorid reaction, while the remainder show practically no change. In all cases the cell count has shown a steady reduction; the globulin content has been more varied, but in most cases has fallen to normal with the improvement of the patient." "We have a number of cases of paresis," writes Barrett, "who were treated intraspinally, in which the diagnosis was unquestionable, who are now carrying on their accustomed occupations with remarkable efficiency, considering their disease."

18. Personal communication to Dr. Riggs.

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Amsden[16] states that "intraspinal treatment with salvarsanized serum has in a considerable number of cases a positive influence in checking the progress of the disease, at least clinically; it does not stop it except in very rare cases." The writer also calls attention to the fact that from the standpoint of the family, the value of this treatment is a matter of importance, since the patient may become able to give information about his affairs, enabling him, as it were, to put his house in order. "In the use of salvarsanized serum," Cotton says, "we have an agent which does cause definite arrest in paresis, which arrest includes improvement in the clinical symptoms, physical signs and a corresponding change in the biological reactions from positive to negative." The writer points out that as it is necessary to treat tuberculosis in the incipient stage in order to obtain results, that it was equally true in regard to paresis, it was fallacious to condemn the treatment of paresis because we could not cure the end stage. He says:

> The emphasis should be placed on treating the cases early. This is especially so in regard to state hospital work, where 75 per cent. of the cases are admitted too late for treatment to be of any benefit to them, but 25 per cent. can be materially improved, as shown by our records. The mortality from paresis has dropped 33⅓ per cent. in the hospital in the last three years. We also have no bedridden paretics, which as a rule are the bane of most hospitals. If these results can be produced on fairly well advanced cases, it augurs well for the success of the treatment of early cases.

The time, then, of greatest opportunity in the treatment of paresis is in its incipiency, before the parenchyma of the cortex has become the seat of degenerative change. "It would be unfortunate," says Amsden, "if apparent poor permanent success in arresting the disease should keep us from trying it and trying to improve it." We are in entire accord with Ogilvie when he says that intraspinal treatment is entitled to

19. Amsden, George: New York Neurological Society, Jour. Nerv. and Ment. Dis., March, 1916.

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recognition as a rational therapeutic procedure and that a far greater percentage of all kinds of cases respond to it than to the older methods of therapy. We have persisted in the use of the Swift and Ellis procedure because there is less hazard in its use, and therapeutically it is as efficient as any. Ayer[20] has described intraspinal injections of serum under prolonged high pressure. He has been able to demonstrate the serum in the subarachnoid space over the frontal lobes. It is possible that this method may possess an increased curative value. Mercurialized serum has given us good results, although it has seemed somewhat more irritating than salvarsanized serum. Several times the injection has been followed by incontinence of urine. We have not employed cerebral puncture as suggested by Wardner;[21] there is much to commend it, and in cases of paresis of long standing it is perhaps the method to be chosen. It is certainly preferable to ventricular puncture as suggested by Hammond & Sharpe. The employment of Ogilvie's standardized serum is a conservative means of injecting salvarsan directly into the spinal canal. Its beneficial effect cannot be doubted. Cotton is using this method for cerebral puncture and mercurialized serum for both cerebral and intraventricular puncture. While the value of drainage of cerebral spinal fluid cannot be explained on theoretical grounds, yet benefit has, in our observation, followed this procedure in some hitherto intractable cases. Sachs believes that he has seen as marked and satisfactory improvement from intravenous as from intraspinal therapy, and he has come to feel that the latter has been unduly pushed; it is more difficult to give, more likely to lead to serious complications, and at times it has been followed by disastrous consequences. Cotton says he considers intra-

20. Ayer, J. B.: Cerebral Syphilis, A Method of Treatment by Prolonged Intraspinal Injections of Serum under High Pressure, Boston Med. and Surg. Jour., clxxiv, No. 17.

21. Wardner, D. M.: Intracranial Injections of Salvarsanized Serum, Tr. Am. Med.-Psychol. Soc., 1915, xxii.

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venous injections of salvarsan more dangerous than intraspinal; that with myocardial trouble the former might prove fatal. He says:[22]

> We have given as near as I can figure about 3,000 treatments in the last three and a half years, and have had no unfavorable results whatever, except one or two cases have developed an irritative meningitis which subsided within a week without any bad results.

Ogilvie states that the use of his serum "in more than 1,800 treatments has shown it to be both safe and effectual as a curative agent in types of syphilitic nervous diseases, in which intraspinal therapy is indicated." We have made over 600 injections of salvarsanized serum (Swift and Ellis method) with no serious ill effects. In one patient only, there developed an aseptic meningitis, which quickly subsided. Our experience teaches us that this mthod is a safe and conservative form of therapy, and should be preferred to intensive medication in all serious cases of cerebrospinal syphilis and its late manifesation, paresis and tabes. Over 74 per cent. of Ogilvie's series of cases were influenced favorably to a degree not attained by the most heroic intravenous treatment, either with or without mercury.

CONCLUSIONS

1. Both clinically and biologically it has been demonstrated that treatment is a material benefit to the paretic.

2. Before the salvarsan era, the number of remissions varied from 4 to 20 per cent. Since its use and the employment of modern forms of therapy, they have been greatly increased in frequency.

3. Treatment, to be effective in paresis, must be given in its earliest stage; in cases of long standing it is useless; cell destruction cannot be replaced. Also, to be effective, it must be persistent. It may be a matter of many months before the activity of the infection can be influenced.

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22. Personal communication to Dr. Riggs.

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A CONSIDERATION OF THE SEROBIOLOGIC REACTIONS AFTER FIVE YEARS OF OBSERVATION

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Somebody has said that the serobiologic reactions have created a new science. It may also be said they have given us a new symptomatology. If we regard them as symptoms and not as reactions, they become much easier of understanding and interpretation. The lack of understanding concerning them has been due chiefly to two things: First, we do not know the source of the so-called antibodies which are necessary for a positive Wassermann, or the origin of the lymphocytosis or increase of globulin content which occurs not only in the spinal fluid but in the blood as well. Naturally, then, all our knowledge thus far in regard to these reactions has been obtained by the inductive method, that is, drawing conclusions from individual observations and striking an average or a percentage to be used in the formulation of general rules about them from the sum total of observation.

In any knowledge obtained in this way, it is natural to expect that new observations will be reported from time to time which require some modification of previous conceptions. In the second place, in the application of general laws to any particular class of cases, there are always the exceptions to be taken into consideration. These reactions or symptoms were often found where, from the nature of the case, we did not expect them to occur, or, on the other hand, absent in cases in which, according to our previous knowledge,

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we thought they ought to be. If, then, we view these reactions as symptoms, which they certainly are, and study their interpretation in connection with and as we do other symptoms in any case, much of this past confusion in regard to them will disappear. We do not expect to find all the symptoms of a disease present in every case. Not every case of meningitis presents a Kernig's sign or rigidity of the neck. Furthermore, not every case of rigidity of the neck is diagnosed as meningitis. Nystagmus is a common symptom of multiple sclerosis, but it would be a serious mistake to regard every case in which the symptoms of nystagmus was present as a case of multiple sclerosis. Tremor is an important and frequent symptom of paralysis agitans, yet we would fail to recognize many cases of this affection if we excluded the diagnosis because of the absence of tremor.

We must learn to think of the serobiologic symptoms as we do all other symptoms and realize that their value and the interpretation which we give them is not an isolated one, but depends largely on the syndrome which accompanies them. In this new symptomatology, as in the old, it is not in the typical cases in which the symptoms are present that the difficulty in the interpretation arises, but in those cases in which these symptoms are irregular or present only in part.

In the beginning of the Wassermann era the impression was quite general that a negative Wassermann made the diagnosis of syphilis extremely doubtful. We are still often confronted with the statement that the Wassermann is negative, as if the final word with reference to syphilis in the case under consideration had been spoken. We know positively at present from records made by hundreds of observers all over the world that in syphilis of all stages a serum Wassermann is found in only about 65 per cent. of the cases. We do not often find it in the primary stage. In the secondary stage it is nearly always present,

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and in the tertiary in perhaps from 40 to 50 per cent. of the cases. In tabes this symptom is present in from 60 to 70 per cent., in cerebrospinal syphilis in 80 per cent., and in paresis in from 95 to 100 per cent. of all the cases. In my own work, in sixty cases of syphilis without symptoms referable to the nervous system, the Wassermann was positive in thirty-nine, or 68 per cent.; in eighty-one cases of cerebrospinal syphilis, in forty-three, or 53.3 per cent.; in fifty-seven cases of paresis, in fifty-three, or 92.7 per cent., and in twenty-six cases of tabes, in thirteen, or 50 per cent. It ought to be perfectly plain, from a perusal of such reports, that in a large number of cases of syphilis the serum Wassermann is absent, so that we are able to say positively that the absence of this symptom in the blood serum proves nothing either for or against the assumption of specific disease.

Observation[1] has shown us that a definite relationship between the activity of the syphilitic processes and the Wassermann reaction, as well as the other reactions in the blood and spinal fluid, does not always exist. Late syphilis, bone syphilis, tabes and cerebrospinal syphilis with extensive involvement and active symptoms may give negative reactions, while, on the other hand, cases with strong positive reactions occur in persons who are apparently in good health. A case of paresis, in a complete remission, may give just as strong a positive Wassermann, or just as much of an increase of the lymphocytes in the spinal fluid as an active and rapidly progressive one. There are numerous cases which, after a mild treatment, lose their positive reactions, only to have them return after a short interval. On the other hand, there are cases which, before the beginning of treatment, are negative, but afterward become positive. In one of my cases, a positive serum Wassermann, after the administration of salvarsan intravenously, became negative

1. Von Wassermann, A., and Lange, C.: Zur Frage des Entstehens der Reaktions producti bei der Serodiagnostik auf lues, Berl. klin. Wchnschr., March 16, 1914.

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and remained so, although the clinical condition gradually grew worse and the patient finally died. In another case, a positive reaction remained positive in spite of vigorous treatment, although the symptoms entirely disappeared. Practically all of the authorities at present regard a positive Wassermann as proof of a syphilis either past or present, and the weight of opinion considers that it is present.[2] In other words, a positive Wassermann is thought to mean the presence somewhere in the system of living spirochetes. The exceptions to this rule are so few, about 0.5 per cent., and the other diseases in which it occurs are so easy to differentiate from syphilis by the symptoms and history, that their importance may be considered negligible.

The spinal fluid reactions, especially in obscure conditions, and where the nervous system is involved, are of much more practical value, in both diagnosis and prognosis, than the serum Wassermann. It is quite the vogue now to have a serum Wassermann made on the patient; but to have a thorough examination of the spinal fluid is comparatively rare. In all cases with obscure symptoms referable to the nervous system, the spinal fluid examination is of vastly greater value. In my work I have been much impressed with the large number of cases in which what may be called the cardinal symptoms of nervous syphilis, such as pupil anomalies, Romberg, absent knee jerks, sensory disturbances, etc., were absent, and the symptoms present were few in number and obscure in character. In this category may be mentioned especially neuralgias and paresthesias of the fifth nerve, sciatica, Bell's palsy, persistent insomnia, symptomatic migraine, and choreiform movements not unlike the type occurring in Sydenham's chorea. In many of such cases the serum Wassermann is negative, and unless a spinal fluid Wassermann is made, only serves to mislead one.

2. Nonne, M.: Syphilis und Nerven System, Ed. 3, p. 813.

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CASE 1.—A man, aged 40, whose only somatic symptom was a twitching of the left cheek and lower eyelid, subjectively had no complaints. Both the physical and neurologic examinations were negative. A history of a syphilitic infection, dating back fifteen years, was obtained. The serum Wassermann was strongly positive. Likewise, the spinal fluid in 0.2 c.c.; the lymphocytes were 80/3; the globulin ++, and the goldsol gave the typical paretic curve. Within six months from the date of this examination, although specific treatment was begun immediately and continued during this entire period in the form of weekly injections of mercuric salicylate, the patient began to get irritable and quarrelsome, his sleep was disturbed, and he soon became violently insane. This case ran a galloping course, and in a little more than a year the patient died of paresis in a state hospital.

This case is interesting for the reason that six months before the appearance of any somatic or psychic symptoms of paresis, how much longer, no one knows, the serobiologic symptoms of paresis were all present. It is further interesting from a therapeutic standpoint because specific treatment was begun at once, and on the first appearance of mental symptoms, salvarsan intravenously and salvarsanized serum intraspinally were administered and then followed with a course of sodium nucleinate injections, but without any result in staying the progress of the disease.

Case 2 illustrates the incongruity often existing between the clinical and serobiologic symptoms. It also shows how misleading a serum Wassermann and the symptoms which we formerly had to depend on for our diagnosis may be, as well as the value of the spinal fluid examination in every suspicious case.

CASE 2.—T., man, aged 45, married, American, salesman, examined March 2, 1916, had one brother who died of paresis and another who had an advanced tabes. The patient had been married seventeen years, and had one child, living and well; there was no history of miscarriages. Fifteen years before he had a chancre and mild secondaries; treatment at that time consisted of yellow mercurous iodid pills for about two years. Since then he had had potassium iodid and some mercurial inunctions at intervals, but had not taken any systematic course after the first two years. The only complaint at the present time was a sensitiveness in the upper

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part of the nose internally and a stuffy feeling in the head, as if he had a cold. These symptoms were not constant. The nose and throat specialist whom the patient consulted could find no explanation for them in the nasal passages. The patient visited me, not because of these symptoms, but for the reason that the death of the brother from paresis frightened him, and he wished to find out if any traces of his former syphilis were still present. The serum Wassermann, a luetin test and the neurologic examination, all resulted negatively. He was then persuaded to have an examination made of his spinal fluid. This gave a strongly positive Wassermann with 0.2 c.c., the minimal quantity of fluid, a Nonne + +, lymphocytes, 172/3, and the goldsol reacted in the paretic zone. Up to the present time the patient's condition continues good.

It is interesting to note that the origin of the infection in all three brothers was different. This argues against the doctrine often expressed of a certain type of syphilis which possesses a special virulence for the nervous system and in favor of the theory of lessened family resistance which is so often observed in other infectious diseases.

In a certain type of cases in differential diagnosis the globulin and lymphocytes reactions have been of more value to me than the Wassermann. In many cases with organic nervous involvement the Wassermann has been absent in both the serum and spinal fluid, even when 1 c.c. of the fluid was used, and the diagnosis had to be made from the history, clinical symptoms and the spinal fluid findings without the Wassermann. In such cases, with either no history or a negative history of specific infection, the variations in intensity of these two spinal fluid findings, carefully considered, in conjunction with the clinical symptoms, have often proved the deciding factors, in making the diagnosis. The following cases are illustrative of this:

CASE 3.—B., aged 26, farmer, seen in consultation Dec. 26, 1915, could give only an imperfect history on account of his mental condition. There was nothing of importance in the family history. It was learned from his father that he smoked and drank to excess, but on the advice of a physi-

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cian had stopped drinking about five months before. In June, 1915, he began to have paroxysms of pain in the region of the left kidney which were thought to be due to gravel. In November, he had a very severe attack of pain lasting several hours. From this time on, at night, he began to whistle and sing, and kept up such a disturbance that his room had to be changed so the other members of the family could sleep. During the day he seemed rational. This condition remained the same until December 4, when he began to have trouble in passing urine. He became unable to void it, and some difficulty was experienced in catheterizing him. At the time I saw him, December 10, he was delirious all of the time, and had involuntary actions of both bladder and bowels. His temperature, although it had been normal for the two days previous to my visit, varied between 101 and 101.6. The pulse ranged between 90 and 112. The patient was rather emaciated and had a slight cough. Heart and lungs were negative. the urine contained albumin and quite a number of red blood cells. The inguinal glands were distinctly enlarged; also, two epitrochlear, about the size of a split pea, were present on each side. There was a general tendency to a coarse tremor. A suggestion of ankle clonus and a positive Babinski on the left side were the only somatic symptoms observed in the neurologic examination. The serum Wassermann was negative. A spinal puncture was made, December 11. On that day the temperature reached 103. The spinal pressure was slightly increased. The spinal fluid was clear; Nonne, +; lymphocytes, 20/3; spinal fluid Wassermann, negative with 0.2 and 1 c.c. of fluid. The goldsol gave what might be called a freak reaction, with no change in the first tube, a reddish blue in the second, blue in the third, and no further changes in the remaining tubes. On the same day of the puncture mercurial inunctions were begun. On the following day, the patient became worse and went into an unconscious condition in which he remained for a week.

In this case the enlarged epitrochlear and inguinal glands, as well as the history of development, with delirium coming on at night and disappearing during the day, led one to suspect syphilis. On the other hand, the emaciation, slight cough, and the high temperature caused one to feel that tuberculosis was also a probability to be reckoned with. From the history and the neurologic examination as well as the spinal fluid findings, it seemed quite certain that the case was a meningitis and that this meningitis was either syphilitic or tuberculous in character. After careful consideration

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of both the reactions and symptoms, tuberculosis was excluded, chiefly because of the mild degree of intensity of the globulin and lymphocyte reaction. The position was taken that in any case of tuberculous meningitis in which the symptoms were so extensive and severe, both the globulin and lymphocyte reactions would be much increased. The mildness of these reactions was thought to be absolutely out of harmony in a tuberculous meningitis manifesting such acute and extensive symptoms. The negative Wassermann in the blood and spinal fluid was given the importance only which should be given to any negative symptoms. We know absolutely that we cannot exclude syphilis because the serum Wassermann is negative, and we must come to the same opinion relative to the spinal fluid Wassermann. If it is absent we must not, for this reason alone, exclude the possibility of nervous syphilis, but merely apply to its absence whatever other differential diagnostic value this absence may possess. It does sometimes have a very particular value, which is well brought out in the next case:

CASE 4.—T., aged 35, merchant, married, with two healthy children, stated that his father died from tuberculous meningitis, which was the significant thing in the family history. The patient himself, five years before, had a tuberculous affection of the lungs which was at this time apparently quiescent. He also gave a history of a specific affection dating back ten years. He seemed in good health up to the time of the present illness. He had been sick two weeks when I saw him. He then complained of a pain in the head and back of the neck. His eyes were very sensitive to light. He was extremely nervous and restless, with no appetite and bowels constipated. His temperature ranged from 99 to 101. The only objective symptom elicited in the physical and neurologic examination was a slight rigidity of the neck muscles. The serum Wassermann was negative. The spinal pressure increased. The spinal fluid Wassermann in 0.2 and 1 c.c. was also negative. The globulin was strongly positive, the lymphocytes 100/3. The goldsol test showed the greatest change in the dilutions from 1:160 to 1:640. In this case the absence of the Wassermann in the spinal fluid taken in connection with the marked increase of the lymphocytes, was very significant. According to our past experience

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all spinal fluids in specific cases with so large a lymphocyte increase had given a positive Wassermann. For this reason it was thought that syphilis could be safely excluded. Accordingly the diagnosis of tuberculous meningitis was made. The subsequent history of the case confirmed this diagnosis.

The Wassermann reaction is, of course, the most reliable of all serobiologic symptoms when present in differentiating between nervous syphilis and other organic disease of the nervous system; but it is so often absent, even in the spinal fluid, and there are so many times when the distinction must be made without it, that I have come to depend a great deal on the globulin and lymphocyte reactions in my work. The lymphocyte reaction has been especially valuable. The diseases in which it occurs with a symptomatology that might be mistaken for syphilis are not numerous, and even then there is usually some factor which in itself is distinguishing, as in the case just reported of tuberculous meningitis, with a negative Wassermann and the large lymphocyte increase.

The normal number of lymphocytes in a cubic millimeter has been often stated to be from nothing to five, with a borderland of nine or ten.[3] According to my observation, I believe this estimation of the normal number of lymphocytes too high. Five lymphocytes to 1 c.mm. would give fifteen in the 3 c.mm. in the Fuchs-Rosenthal counting chamber, while the usual count in normal cases for the whole space is certainly much less than this. In the following case a pupil anomaly and the lymphocyte count were the most important differentiating symptoms:

CASE 5.—G., man, aged 40, clerk, unmarried, had been sick about four weeks when he came under my care; he was then incoherent, disoriented as to time and place, and unclean in his habits, with an inclination to expectorate on everybody and everything near him. He complained of some headache, which was increased by coughing or sneezing. His appetite was excellent, his mental state, one of exhilaration and good humor, his memory, very poor. On account of the mental condition, no past history or history of a present illness was obtainable. Physically, his condition was good.

3. Nonne, M.: Syphilis und Nerven System, Ed. 3, p. 99.

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Pulse and temperature were normal; urine examination was negative. Station was bad, and when he attempted to walk, he staggered like a drunken man. He did not lie still for a second when awake, but kept twitching and jerking, like a person with an aggravated case of chorea. The right pupil was larger than the left, but both reacted to light. No other pupil anomalies were observed. The patient's articulation was good, but he was coarse and loud in his talk. Spinal pressure was not increased. Both the serum and spinal fluid Wassermanns were negative. The globulin reaction was also negative and the lymphocyte count was 18/3, just one more to the cubic centimeter than is given as normal. Brain tumor or cerebrospinal syphilis seemed the most probable conditions to be considered. However, on account of the number of lymphocytes, the latter diagnosis was made. This patient made a good recovery with mercurial inunctions and intravenous injections of salvarsan. After his recovery a history of specific infection of twelve years' duration was obtained.

When even a slight increase in the number of lymphocytes is often so important in deciding the diagnosis, the necessity for obtaining the spinal fluid absolutely free from blood can be readily appreciated. The faintest trace of it will give a positive Nonne and a lymphocytosis, and if not detected, lead to erroneous conclusions.

The study of these reactions in the spinal fluid was at first almost entirely confined to patients manifesting nervous symptoms. Recently, however, a great many observations on their behavior in the spinal fluid in primary and secondary cases of syphilis without any nervous symptoms have been made. M. Fraenkel[4] was among the first in reporting on the spinal fluid reaction in fifteen cases of untreated secondary syphilis, without evidence of nervous involvement. He found the Wassermann positive in five of these cases. Dreyfus and Assmann, in a large number of examinations in secondary syphilis, found that the spinal fluid was normal in only 22 per cent. of the cases. In later examinations on the same patients, 75 per cent. of them gave negative reactions.

4. Fraenkel, M.: Weitere Beiträge zur Bedeutung der Auswertungsmethode der Wassermann-reaktion in Liquor cerebrospinalis, Ztschr. f. d. ges. Neurol. u. Psychiat., xi, Nos. 1. 2.

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Wile and Stokes,[5] in an article on the spinal fluid in secondary syphilis, conclude that from 60 to 70 per cent. of all cases which progress to the secondary stage show some change in the fluid, which indicates a nervous involvement. In a later article on the same subject they say: "We now feel our figures in our former article were too conservative and in all probability every case of secondary syphilis has an involvement of the nervous system." A consideration of these observations and others similar in character indicate to what extent the study of these reactions have changed our conceptions of syphilis and especially the syphilitic invasion of the nervous system. A nervous syphilis has always been regarded as belonging distinctly to the later manifestations of that disease. In the light of these recent investigations we may now believe that the nervous system shares in the general invasion of the body during the primary and secondary stages. In the great majority of these cases either as a result of treatment or a naturally developed resistance, recovery takes place and the spinal fluid becomes normal. Dreyfus has ascertained that in approximately 12 per cent. of the syphilitics, without nervous symptoms, examined by him, the spinal fluid has continued pathologic. He compares this percentage with the 12 per cent. of specific organic nervous affections found by Mattauscheck and Pilcz among 4,000 army officers who had contracted syphilis. In commenting on this comparison, Nonne[6] says that the conclusion lies near that these cases in which the fluid remains pathologic are the ones which later on develop the syphilogenic nervous affections. The conception at present of the development of a specific nervous disease is then a latency, dating back to the primary and secondary stages, which continues latent until some exciting cause transforms the latent process

5. Wile and Stokes: A Study of the Spinal Fluid, with Reference to the Involvement of the Nervous System in Secondary Syphilis, Jour. Cutan. Dis., September, 1914, p. 607.
6. Nonne, M.: Syphilis und Nerven System, Ed. 3, p. 139.

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into an active one. Clinical experience is also in harmony with this view. In one of my cases, a cerebrospinal syphilis developed several weeks after a severe head injury. The patient was apparently well before his accident, but soon afterward he complained of a severe headache and became mentally very dull. He also developed a hemiparesis on the right side. The spinal fluid gave an increased globulin and lymphocyte reaction. These symptoms entirely disappeared with specific treatment.

In regard to head injuries, Nonne says, "It has been known for a long time that they can cause an outbreak of brain syphilis, and especially the development of gummatous brain tumor has been observed following such injuries." It is not rational to suppose in these cases that the nervous tissue became infected with spirochetes after the injury from some distant focus. The only logical explanation is that these germs were already present in the injured tissues in a latent state, and the injuries so lessened the normal tissue resistance that the latent process was converted into an active one.

In prognosis, as has been stated, and as we have seen in the cases reported, neither the number of the reactions present nor their degree of intensity bears any direct relationship to the rest of the symptomatology. In Case 3 the syphilitic process was an exceedingly acute and progressive one, yet the only reactions present were the globulin and lymphocyte, and these were mild in character. On the other hand, in Case 2 there was entire absence of subjective and somatic symptoms and yet the reactions were all intensely positive. Case 1, with a slight twitching of the face as the only somatic or subjective evidence of nervous disease, in which all the reactions were strongly positive and which, in a short period of time developed into a galloping paresis, would seem to point to a gloomy prognosis when such conditions exist. Nonne has reported in this connection thirteen

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cases in which the reactions were positive, both in the blood and in the spinal fluid. Three of these cases were progressive in character; the other ten remained stationary over a period of five years. The following case is quoted from Nonne:[7]

A man, aged 62, formerly a captain, has been a tabetic for fifteen years. Mercurial treatment, twelve years ago. Since then no specific treatment. The pupils do not react to light. Knee jerks are absent. Hitzig's zone. Four years ago Wassermann in blood, + + +. Wassermann in spinal fluid, + + with 5 c.c. of fluid. Globulin, +; lymphocytes, +. At present practically no change in either the reactions or somatic findings. The patient feels well and works every day.

This case is not an isolated case, but one of a series of cases which Nonne has observed.

The next case is a report on the reactions in an old case of tabes, the first symptoms of which appeared about twenty years ago:

Case 6.—This case has gradually progressed in a halting way during all of these years and is still advancing. The chief discomforts now are a high degree of ataxia, lightning pains which are exceedingly severe in character, and loss of bladder control. Feb. 15, 1915, the serum Wassermann was slightly positive; spinal fluid examination pressure, normal; globulin, negative; lymphocytes, 3/3; goldsol, typically in the syphilitic zone; spinal fluid Wassermann, negative in 1 c.c. March 1, 1916, a little over a year later, the serum Wassermann was negative; spinal pressure, normal; globulin, negative; lymphocytes, 6/3; spinal fluid Wassermann, negative; goldsol, still in the syphilitic zone. Except for the goldsol, the spinal fluid reactions, at both examinations, might be considered normal, yet during the period between these examinations, this patient has been practically bedridden because of the high degree of ataxia and has suffered most severely from lightning pains.

When such contradictory observations as I have cited are compared, the whole subject of the interpretation of these reactions from the standpoint of prognosis seems to be in confusion. The confusion, however, is not so complete as it appears. Many cases of cerebrospinal syphilis of the genuine type have been reported in which the spinal fluid became normal,

7. Nonne: Syphilis and the Nervous System, Ed. 3.

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either spontaneously or as a result of treatment. In these cases, so far as I know, there have been no relapses reported. On the other hand, observations thus far indicate that cases in which there is a pathologic fluid are never safe from relapsing. It seems quite plain that we cannot consider any case of syphilis as having recovered, and this applies as much to primary and secondary syphilis as it does to the nervous type until the spinal fluid becomes normal. Absence of symptoms and repeated negatives in the blood cannot be regarded as recoveries in those cases in which there is still a pathologic spinal fluid. For this reason no patient should be dismissed by the syphilologist and treatment stopped without a complete spinal fluid examination. In the so-called cases of parasyphilis, such as tabes and paresis, the outlook for obtaining normal spinal fluids, with present methods of treatment, except in the old *abgelaufene* type (cases in which the disease has spent itself), such as the last case reported, as rather hopeless, and in treating these cases this should be realized.

The variations in intensity of the reactions in such cases or the changing of some of the positives into negatives as a result of treatment have, in my opinion, very little value from a prognostic standpoint. The following explanation of the reactions in the type of cases represented by Cases 2 and 3, in which such an incongruity exists between the reactions and the symptoms, is suggested. In Case 3 the process is an active one and the invasion of spirochetes is limited in extent. In other words, it is an active syphilitic process of recent origin and with a good prognosis. In the type represented by Case 2, the process is an extensive one. The whole central nervous system is invaded, but up to the present time it is latent. Sooner or later something will occur to make it active, and then the course will be rapid and severe, as it was in Case 1, in which the conditions were somewhat similar.

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CONCLUSIONS

The serobiologic reactions should be regarded in the same manner as the symptoms in a clinical picture. They furnish only a part of the information, which must be carefully weighed in connection with all of the other facts obtained. Often when other symptoms fail, they are found to be present, and give the clue to the proper diagnosis. On the other hand, we must not forget that the reverse is sometimes true and they are absent, and then we must depend on the other symptoms for our diagnosis. In atypical cases, hard and fast rules cannot be formulated for these reactions any more than for the other symptoms.

A spinal fluid examination should be made in all cases in which symptoms are present which are referable to the nervous system, unless the etiology of these symptoms is definitely known.

An examination of the spinal fluid is the surest means we have at present of discovering the presence of latent syphilis, and in all cases of syphilis before the patients are discharged from further treatment, this ought to be done, in both early and late cases.

In the absence of other symptoms, its findings form the best criteria in deciding when it is safe to stop treatment. In the so-called cases of parasyphilis the last statement does not apply because of the impossibility with the present methods of therapy of obtaining a normal spinal fluid.

In nervous syphilis, without other symptoms, a normal spinal fluid means a good prognosis and a recovery; a pathologic one, a latency which at any time may become active.

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MERCURIAL MEDICATION WITH SPINAL DRAINAGE

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This is intended to be a brief paper, and few references will be given. The literature of syphilis of the nervous system is now so extensive that it properly falls into various categories, though sharp divisions are not always made into clinical, serologic, etc. We seem to be no nearer a valid line of distinction between paresis and cerebrospinal syphilis than we were, and most authors are becoming very cautious in their assertions as to what does and what does not constitute paresis. There is no reason to suppose that there is any essential difference in the spirochetes in these two conditions, but there is experimental evidence to sustain the view that the spirochete which attacks principally the brain is somewhat different from the one which cripples the heart or assails the liver, as witness a recent communication from the Army Medical School to Dr. Archibald Church.[1]

On anatomic grounds, Dunlap[2] characterizes general paralysis as "a subacute or chronic inflammatory and degenerative process which goes through and through the great central nervous organs," whereas "the varieties of cerebral syphilis on the other hand, for the most part, play around the surface of the central nervous system, instead of going all through it." In making a serologic distinction, Kaplan[3] considers the gold-sol test of more value than the Wassermann reaction,

1. Church, Archibald: Some Modern Views of Syphilis of the Nervous System, THE JOURNAL A. M. A., May 20, 1916, p. 1596.
2. Dunlap, Charles B.: The Pathology of General Paralysis, Am. Jour. Insan., October, 1914, p. 249.
3. Kaplan, D. M.: Serology of Nervous and Mental Diseases, Ed. 1, 1914, p. 191.

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and of course the findings in any case of nervous syphilis may have been altered by treatment. Kolmer[4] says, "In cerebral syphilis the blood serum, and particularly the cerebrospinal fluid, will react positively less frequently than in general paralysis"; but this is doubtful. It is sometimes said that the cell count runs higher in cerebrospinal syphilis, but the largest count which I have seen, 360 cells, occurred in the fluid of a patient with undoubted paresis. Darling and Newcomb,[5] in four of five cases of cerebrospinal syphilis, failed to find more than six cells, and in a fifth case never found more than 100. They lay stress on the great variation in cell count in these conditions but state that it was no greater in the one condition than in the other. They, with Weston,[6] had previously reported three cases of cerebrospinal syphilis in which the cell count never exceeded 3 per cubic millimeter. Two of the cases now being reported have never shown to exceed 5 cells per cubic millimeter, and one has varied from 130 to 360, all being cases of paresis.

There seems to be no reason for making a distinction in treatment between paresis and cerebrospinal syphilis, and no extravagant hopes can be built on any form of treatment as yet devised in well developed cases of either condition, though prolonged remissions, even in general paralysis, may be expected with vigorous treatment in early cases. Gilpin and Earley[7] assume that mercury and arsenic given by any of the ordinary routes fail to reach the cerebrospinal fluid, but this is probably not invariably true, as very small amounts of arsenic have been reported. For example, Barbat[8] found 1 part per hundred thousand of metallic

4. Kolmer, John A.: Infection, Immunity and Specific Therapy, Ed. 1, 1915, p. 462.

5. Darling, Ira A., and Newcomb, Philip B.: The Correlation of Clinical and Serological Findings in Paresis and Cerebrospinal Syphilis, Am. Jour. Insan., January, 1916, p. 449.

6. Weston, Paul G., Darling, Ira A., and Newcomb, Philip B.: The Colloidal Gold and other Tests Applied to the Spinal Fluid in Psychiatry, Am. Jour. Insan., April, 1915, p. 773.

7. Gilpin, Sherman F., and Earley, Thomas B.: Drainage of Cerebrospinal Fluid as a Factor in the Treatment of Nervous Syphilis, THE JOURNAL A. M. A., Jan. 22, 1916, p. 260.

8. Barbat, J. H.: Finding of Arsenic in Cerebrospinal Fluid Following Intravenous Administrations of Neosalvarsan, California State Jour. Med., 1915, xiii, No. 11; abstr. THE JOURNAL A. M. A., Dec. 18, 1915, p. 2199.

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arsenic in the fluid immediately following the administration, presumably intravenous, of a full dose of neosalvarsan. Gilpin and Earley ask, "Is it not reasonable, then, to think that if we reduce the pressure within the cerebrospinal cavity we may succeed in bringing foreign substances through the capillary walls and thus secure the direct effect of drugs such as arsenic and mercury on the nerve tissue itself? The plan carried out on three patients reported at that time was repeated lumbar puncture, performed every week or every two or three weeks, together with mercurial inunctions. Paresis, taboparesis and tabes were represented, and the results were quite encouraging.

On theoretical grounds it does not seem that the removal of from 20 to 40 c.c. of fluid once every week or two should have more than a very transient effect in reducing pressure, and it seems probable that spontaneous variations in pressure may occur from time to time, though doubtless in less degree. The fluid is probably rapidly replaced after withdrawal, and it is possible, of course, that the very rapidity might have a tendency to carry over foreign substances which happened to be circulating in the blood at that particular time. At any rate, the method seemed worth trying, and ten cases of paresis, taboparesis and cerebrospinal syphilis were so treated, that is, the patients were given mercurial inunctions, 1 dram six days in the week, and were punctured every two weeks with few variations. In most cases the inunctions were continuous. They were not selected patients, but would represent about the usual hospital run, and some would be a severe test for any form of treatment. The amount of fluid withdrawn varied from 20 to 35 c.c., the Wassermann was performed with 1 c.c. of fluid by Dr. Clarence Emerson, and the cells were counted in a Thoma-Zeiss chamber with the high power. On the whole, the results are not striking, and similar effects might reasonably be expected without spinal drainage. One of the patients is decidedly better and several are slightly

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so, but the one who improved the most had an alcoholic as well as a syphilitic factor. None have been treated in this fashion for more than about four months, and none for less than ten weeks, and this is perhaps not time enough for a maximum amount of benefit.

REPORT OF CASES

CASE 1.—White man, aged 35, laundryman, married, no children, always intemperate, admitted Jan. 10, 1916, acquired syphilis at the age of 19, and has had gonorrhea several times with resulting stricture. He had a little treatment following chancre, but probably not very much. He first showed signs of mental disturbance in July, 1915, after drinking heavily. He was excited, nervous, sleepless, suspicious and threatening.

TABLE 1.—CEREBROSPINAL FLUID IN CASE 1

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/10	++	++	.....	40	25	++++
3/24	++	++	+	24	30	++++
3/10	++	++	+	34	35	++++
3/24	++	++	+	?	30	++++
4/ 6	++	++	+	31	30	++++
4/20	+	++	+	30	35	++++
5/ 4	+	+	++	16	30	++++
5/18	+	+	+	27	35	++++
6/ 1	+	+	+	28	25	++++

He spent ten weeks in a general hospital before commitment, and received specific medication, including two intraspinal treatments. On admission he was found well oriented, with little disturbance of conduct aside from irritability. Right pupil was large and rigid to light, left moderately responsive to light and distance. Knee jerks were exaggerated, cremasteric, abdominal and gluteal reflexes absent. Slight muscular unsteadiness was present. The Wassermann reaction was positive in blood serum and later in spinal fluid (Table 1). A diagnosis of cerebral syphilis was made. At present the right pupil is large and fixed, while the left reacts only a very little to light. The knee jerks are about as they were, and there is about the same amount of muscular unsteadiness. The patient is fairly reasonable in his conduct, and cooperates well in treatment.

CASE 2.—White man, aged 52, carpenter, wife dead, three children, admitted Sept. 28, 1915, denied intemperance and venereal infection. Trouble had been steadily progressing for about two and one half years, ataxic gait, speech, etc.,

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and delusions that people were stealing his tools. On admission the patient showed disorientation, impairment of memory and judgment and slight euphoria. Argyll Robertson pupils were present, knee jerks absent, speech was defective and writing impossible. On account of extreme ataxia, the patient was kept in bed. Blood serum Wassermann was reported negative, Oct. 12, 1915. A diagnosis of taboparesis was made. Spinal fluid later was positive (Table 2). This patient has not improved in any essential respect under treatment.

CASE 3.—White man, aged 55, farm laborer, divorced, three children, admitted Jan. 11, 1916, acknowledged alcoholic intemperance, but denied venereal infection. At time of admittance he was confused, disoriented, stupid and apparently demented, but later much improved mentally. The left pupil was large and fixed, the right reacted to light and distance. Knee jerks were a little exaggerated, cremasteric and

TABLE 2.—CEREBROSPINAL FLUID IN CASE 2

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/15	++	++	.....	35	15	++
2/29	++	++	++	37	25	++++
3/14	++	++	++	40	25	++++
3/28	+	++	+	23	25	+++
4/12	++	++	++	46	25	++++
4/26	+	+++	++	24	20	++++
5/10	+	++	++	24	25	++++
5/24	+	+	++	39	22	.....

abdominal present, gluteal absent, no speech defect. A provisional diagnosis of alcoholic insanity was made, after spinal fluid findings, changed to cerebrospinal syphilis with alcoholism. January 21, Wassermann was positive in blood serum; February 17, doubtful in spinal fluid. Patient is better mentally and cell count has descended from 223 to 38 per cubic millimeter.

CASE 4.—White man, aged 43, salesman, admitted Jan. 21, 1916, married, two children living, one miscarriage at six months, one seven months, fetus which lived a few hours, one child, the first, which died at 12 days and another which died at 10 years, having abnormally large head and deformed lower extremities; presented a history of intemperance and of gonorrhea, but none of syphilis. Three weeks before commitment, the patient complained of sudden feeling of distress on lifting a heavy weight, and soon afterward became increasingly talkative and affirmed that an uncle in Chicago had left him $100,000. He said he was well and his wife was ill and that he had perfected a scheme to keep his balance in the

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bank the same without making fresh deposits. When admitted, patient was a little euphoric and showed defect of judgment, but not of orientation and apparently not of memory. The right pupil was slighly larger than the left, but both were rigid and very small, even in absence of light. There was central dimness of vision in the left eye field, described as a misty appearance. There was history of probable meningitis at the age of 3, followed by squint which later disappeared.

TABLE 3.—CEREBROSPINAL FLUID IN CASE 3

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/17	+	++	.....	223	20	+—
3/ 2	+	++	+	123	20	+—
3/16	+	+	+	120	25	—
3/30	+	+	+	120	25	++
4/13	+	+	+—	68	25	+
5/ 1	+	+	+	73	30	++++
5/15	+	+	+—	64	25	++++
5/29	+—	+	+	38	30	.....
3/16	.....	.....	.....	Blood serum		++++

TABLE 4.—CEREBROSPINAL FLUID IN CASE 4

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/19	+	++	.....	72	25	++++
3/ 4	+	++	+	63	30	++++
3/18	+	++	++	60	25	++++
4/ 1	+	+	++	70	32	++++
4/15	+	+	++	77	35	++++
4/28	+	+	+	50	30	++++
5/12	+	++	+	57	35	++++
5/26	+	++	+	57	25	.....
6/ 9	+	++	++	91	25	++++
3/18	.....	.....	.....	Blood serum		+++

Knee jerks were present; cremasteric, abdominal and gluteal reflexes absent. There was no ataxia, no marked tremor or unsteadiness, no speech or writing defect. March 18, blood Wassermann was +++. A diagnosis of paresis was made. In a general way the patient certainly shows some improvement, that is, he is more reasonable, his delusions are less in evidence, and he has regained much of the flesh that he had lost; but there is no marked change in the spinal fluid.

CASE 5.—White man, aged 48, cigar maker, married, one child aged 20 years, admitted Oct. 25, 1915, gave a history of probable chancre occurring a good many years before. There

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was mention in the physician's return of sexual abnormality for ten years, the exact nature not known. The symptoms began about July, 1915, with sleeplessness, restlessness, later hallucinations of hearing and delusions of plotting. There was no marked alcoholic factor. When admitted, the patient showed no disturbance of orientation, but had delusions of influence and acknowledged hearing voices, especially at night. He was good-natured, but not euphoric. He had a noticeable speech defect and a little defect in writing. The knee jerks were slightly exaggerated, but pupils normal, cremasteric and abdominal reflexes present. He was paroled from November 24 till January 16. The blood Wassermann was positive, March 18. A diagnosis of paresis was made. The spinal fluid Wassermann was variable, and never more than 5 cells

TABLE 5.—CEREBROSPINAL FLUID IN CASE 5

Date, 1916	Globulin (Noguchi)	Albumin (Trichlor-acetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wasser-mann, 1 c.c.
2/19	+	+	.....	5	30	—
3/ 4	+—	—	—	1	30	++++
3/18	+—	—	—	4	25	—
4/ 1	+—	—	—	2	20	—
4/15	+	+—	—	4	22	+++
4/28	+	—	—	4	25	++++
5/12	+	—	—	1	25	++++
5/26	+—	+—	—	1	20	.....
6/ 8	+—	+—	—	2	25	—
8/18	.....	.....	.....	Blood serum		+

were present. The physical condition is essentially unchanged; hallucinations and delusions are less prominent, and efficiency is increased. The patient takes care of the clothing room in the ward in which he resides.

CASE 6.—White man, aged 44, laborer, divorced, two children, admitted Sept. 18, 1915, gave a history of nervousness, speech difficulty and mental confusion for about three months before commitment. When admitted he was quiet, a trifle confused, without definite or systematized delusions. There was no marked disorientation, but some impairment of memory as evidenced by inconsistent statements. Alcoholic intemperance and venereal infection were denied. The pupils were slightly unequal and very sluggish to light, knee jerks exaggerated, cremasteric and abdominal reflexes present, movements and station a little unsteady, and speech and writing defective. February 23, the blood Wassermann and also the spinal fluid were ++++. A diagnosis of paresis was made. The number of cells has been quite variable, but there has been little variation in the intensity of the Wassermann reaction. The pupils are now almost rigid to light, knee jerks

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very much exaggerated, tremor and unsteadiness greater than at time of admittance. The patient cannot be said to have shown the least improvement under treatment.

CASE 7.—White man, aged 38, telephone man, married, who had had no children, and whose wife had had no miscarriages, who was not intemperate and who denied venereal infection, admitted Aug. 22, 1914, was well oriented, his memory was good, no delusions were present, but his condition was not fully appreciated. The pupils were equal, but rigid to light.

TABLE 6.—CEREBROSPINAL FLUID IN CASE 6

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/22	+	+++	+	110	30	++++
3/ 8	+	+++	+	212	25	+++
3/22	++	+++	++	170	35	++++
4/ 6	++	+++	++	180	30	++
4/20	+	+++	+	140	30	++++
5/ 4	++	++	++	156	30	+++
5/18	++	++	++	90	30	++++
6/ 1	+	++	++	130	30	++++
2/22	.....	. ....	.....	Blood serum		++++

TABLE 7.—CEREBROSPINAL FLUID IN CASE 7

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/24	+	++	++	52	25	++++
3/ 9	+	++	++	41	25	—
3/23	+	+	++	33	25	++++
4/ 6	Specimen contained blood		.....	50*	35	++++
4/20	+	++	++	60	20	++++
5/ 4	+	+	++	40	20	++++
5/18	+	+	+	60	25	+++
6/ 1	+	+	+	130	25	++++

* Other than red cells.

Knee jerks present, also abdominal, cremasteric and gluteal reflexes. There was considerable tremor, and well marked ataxia. There was considerable increase of globulin and albumin, and 115 cells per cubic millimeter in the spinal fluid. A diagnosis of paresis was made. Mercurial inunctions and potassium iodid were ordered. There was a mild paretic seizure; Oct. 14, 1915. The number of cells has varied considerably, but the number was high at the last count, and the Wassermann still strongly positive. The pupils are still rigid, the left knee jerk much exaggerated, and the patient is more

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tremulous and unsteady now than he formerly was. He still does not appreciate his actual condition.

CASE 8.—White man, aged 47, tailor, married, three children, admitted Nov. 20, 1915, had no history of intemperance or venereal infection. About two years before commitment his gait became peculiar and he began to drop things. He became very nervous and so forgetful that he could not go on a simple errand and bring back what was wanted. About one year before commitment, spinal fluid was negative. When admitted, patient was disoriented and showed considerable mental confusion, impairment of memory, judgment, etc. The pupils reacted to light and distance, the knee jerks were a little exaggerated, and there was slight defect in speech, writing and gait. A diagnosis of paresis was made. December 6, the spinal fluid showed a slight increase in globulin, but there

TABLE 8.—CEREBROSPINAL FLUID IN CASE 8

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
2/29	+	+	—	0	30	+++
4/ 1	+	+	+	1	25	—
4/15	+—	+	—	4	25	—
4/28	—	+—	—	0	22	++++
5/12	+	+—	—	1	30	++++
5/26	+	+—	—	1	25	.....
6/ 9	+	+	—	8	30	+
1915						
12/ 6	+	+—	.....	4	25	—(Blood serum—)

were only 4 cells per cubic millimeter, the Wassermann reaction was negative in both blood and spinal fluid. Since then the spinal fluid Wassermann has been positive three times and negative twice, and the number of cells has never exceeded four. There has never been more than a slight increase of albumin and globulin, and the Ross-Jones test was positive only once. The knee jerks are now more exaggerated than they were, while tremor and muscular unsteadiness are more pronounced.

CASE 9.—White man, aged 40, laborer, single, with history of intemperance, but none of venereal infection, said to have delusions of God directing his movements, etc., when admitted, Oct. 11, 1914, had ideas of grandeur and unlawful detention, ideas of reference and hallucinations of sight and hearing. The pupils reacted moderately, knee jerks were exaggerated, abdominal and cremasteric reflexes were present and gluteal absent. Later on, disturbance of speech appeared. A diagnosis of paresis was made. Blood Wassermann was positive when admitted. The spinal fluid Wassermann is still + + + + and the patient is no better physically, though he

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is more tractable than he was at one time. June 8, the spinal fluid contained more cells than at any previous time, 360 per cubic millimeter.

CASE 10.—White man, aged 36, painter, single, formerly intemperate, admitted March 14, 1916, one year before commitment had an attack of mental disturbance which lasted about three weeks. Shortly before commitment he became excited, sleepless and confused, with hallucinations and illusions. When admitted, patient acknowledged having had both gonorrhea and syphilis at the age of 18, but said he was

TABLE 9.—CEREBROSPINAL FLUID IN CASE 9

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
3/14	+++	+++	+++	290	25	++++
3/28	++	+++	++	210	28	++++
4/12	++	+++	++	130	30	++++
4/26	++	++++	+++	277	25	++++
5/10	+	+++	+++	208	28	++++
5/24	+	+++	+++	199	30	.....
6/ 8	+++	+++	+++	360	30	++++

TABLE 10.—CEREBROSPINAL FLUID IN CASE 10

Date, 1916	Globulin (Noguchi)	Albumin (Trichloracetic Acid)	Ross-Jones Test	Cells	Amount, c.c.	Wassermann, 1 c.c.
3/16	+	+	+	46	30	++++
3/30	+	+	+	20	25	++++
4/13	+	+	+	17	25	++++
5/ 1	+	+	+	80	25	++++
5/15	+	+	+	85	30	++++
5/29	+	+	+	56	26	.....
3/16	.....	.....	.....	Blood serum		++

treated and cured. Delusions were present and some mental confusion. Pupils were sluggish to light, knee jerks exaggerated, tremor present, speech and writing a little defective. The Wassermann reaction was ++ in the blood and ++++ in the spinal fluid. A diagnosis of paresis was made. The fluid Wassermann was the same at all examinations since admission. Cells have varied from 17 to 85 per cubic millimeter. The right pupil is now a little larger than the left and decidedly sluggish to light. Otherwise the patient is about the same, except that he is steadier and more efficient in his work.

I am indebted to Dr. B. F. Williams for permission to report these cases, and to Dr. Clarence Emerson for the performance of the Wassermann tests.

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ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. SCHALLER, RIGGS AND HAMMES, BALL AND PILSBURY

DR. J. HENRY BARBAT, San Francisco: During the past two years I have been doing experimental work regarding the permeability of the meninges to drugs. Patients were given salvarsan, neosalvarsan, and later arsenobenzol intravenously, and immediately after the injection as much spinal fluid as possible was withdrawn by lumbar puncture, usually as much as 60 c.c. The second half of this fluid was tested for arsenic. Twenty-four hours after the first tapping, 20 c.c. of spinal fluid were withdrawn. Within one hour after the injection of arsenobenzol, 20 c.c. of blood were taken and the serum separated from the clot. These specimens were all sent to the Hygienic Laboratory at Washington to be tested for arsenic. About four fifths of the arsenic is fixed in the body cells within one hour after its intravenous administration, leaving only one fifth in the blood, a proportion of about 1-100,000. With neosalvarsan we have a slightly larger proportion of arsenic getting in the spinal fluid than with arsenobenzol. In my series of cases the analyses showed an average of 10 per million arsenic in the blood, largely in the serum. In the spinal fluid drawn 24 hours after the administration of the drug from a trace to 10 per million, average ¼ per million. The second half of the fluid drawn immediately after the injection of arsenobenzol showed traces of arsenic in 40 per cent. of the specimens. Carr and Hall demonstrated arsenic in the spinal fluid after the administration intravenously in a very small percentage of cases. J. J. Viton of Buenos Aires showed clearly that the permeability of the meninges depends on whether or not they are irritated or inflamed. The injection of homologous serum into the spinal canal will produce leukocytosis; injection of horse serum will produce leukocytosis plus pain and fever. Therefore irritation of the meninges, whether by chemicals or other means will increase the permeability. This brings us to the questions as to whether the intraspinal injection of any drug has any specific effect in the treatment of any of the cerebrospinal syphilitis lesions. I believe that the effect of all these intraspinal injections is to set up a meningitis which, by increasing the blood supply permits the passage of antibodies and drugs into the spinal fluid enabling them to act more efficaciously on the spirochetes. This condition can more easily be brought about by the technic which I spoke about at first.

DR. TOM A. WILLIAMS, Washington: One feature of syphilitic meningitis which is so frequently forgotten is the fact that in the tertiary phase, at least, it is not a serositis; the inflammatory condition is not one of the surface of the membrane, but of the deeply seated blood vessels in the membrane. The lymphocytotic reaction is a mere epiphenomenon, so to

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speak, quite different from the leukocytotic reaction in epidemic meningitis or in pneumococcic meningitis. Therefore, in order to affect the disease one must attack the perivascular lymphatic spaces in the meninges. What Dr. Barbat has just said is, I think, very true. Three years ago I considered the real elements of advantage in intraspinal medication. I need not repeat what I said in view of what Dr. Barbat said. Sicard, at the very beginning of salvarsan therapy, made a practice of injecting intraspinally weak salts of mercury while at the same time mercurializing or salvarsanizing the patient, with exceedingly gratifying results, which are not inferior to those which have been so often vaunted as peculiarly efficient by persons using salvarsanized serum. I believe that the meningeal reaction produced by the introduction of foreign substance intraspinally is responsible for any advantage there may be, although we do not really know that there is any permanent advantage in intraspinal medication, which must always be combined with systemic medication, whether by salvarsan, preferably, or by mercury as was formerly the case. I do not presume to speak of the problem of paresis, for it would take much longer than five minutes to even lay down the ground for it. I am talking only of the syphilitic meningitis which eventuates in what is called tabes dorsalis.

Dr. Albert E. Sterne, Indianapolis: In the last seven years I have seen no reason to change the estimation of the results of treatment of any form, in cases of the kind under discussion. I desire to place particular emphasis on the fact that no neurologic examination is complete without a thorough bioserologic survey of each individual case, for undoubtedly the biologic findings guide us correctly, to my mind not only diagnostically, but, still more important, prognostically. But so far as the ultimate results of treatment are concerned, I have not been able to convince myself that there is any marked gain. We have, possibly, a better temporary result than was formerly secured. But the crux of this whole question seems to me to lie in two directions: First, we must not fail to recall that we achieved excellent results in the vascular forms of syphilis, not only of the nervous system, but of the body as a whole, but particularly of the nervous system, by the methods employed previous to the use of salvarsan. Some of the forms of vascular syphilis affecting the nervous system recovered then as now. The results of treatment of parenchymatous syphilis are hardly better now than formerly. The happy aspect of the present methods of treatment is this: Personally, I believe that every nervous system is affected early after the primary infection. If the patient has headaches within the first few weeks following primary infection, his spinal fluid should be examined, when in most instances it will be found to bear the characteristics of an invasion of the central nervous system. That is the time, if any time, when we

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might expect to accomplish something, not only positively curative, but also prophylactically for the prevention of the tertiary degenerative changes in syphilis of the nervous system. That is the time to give intraspinal medication. I do not care what form you use, whether arsenical or mercurial preparations. It has been our habit recently to use the drainage method, but in a slightly different way. When we give an intravenous injection we immediately follow it with a spinal puncture, injecting nothing into the spine, but withdrawing spinal fluid, and creating, so to speak, a wave toward the cerebrospinal axis. Until we know positively the direction and extent of the flow of the cerebrospinal fluid, we cannot definitely say how much may be gained in this way. Owing to the characteristics of the choroid plexus we can not say, with any degree of assurance, how much or little can be attained, but there is evidence pointing to the fact that we have rapid regeneration of the fluid, and furthermore, that at least part of the spinal fluid is derived from the blood. With the influx into the cerebrospinal axis we have reason to believe that whatever drugs we place in the blood current will soon reach the cerebrospinal axis after lumbar puncture. The time to give intraspinal medication is early, and intravenous medication followed within twenty minutes by spinal puncture would seem to be worthy of trial, in place of direct intraspinal application.

Dr. Julius Grinker, Chicago: I would like to ask Dr. Schaller if the presence of the knee jerks in 13 of the 35 cases was really controlled by him, or was it only reported to him by his interns? It has been my experience that interns will occasionally report the knee jerks as being present in tabetics, when there is only a pseudo-reflex present, which is often found in tabetics, and which is merely a voluntary response. If he has controlled the findings then the percentage of present reflexes in tabes is rather high in his service. My own observation of hundreds of cases of tabetics gives me a smaller percentage of present tendon reflexes. As regards the Achilles jerk, it has been my experience that this reflex is very often lost before the knee reflex. Referring to the negative Wassermann findings in the spinal fluid of eleven cass of tabes, I wish to ask Dr. Schaller whether they were not advanced cases of tabes. I have in mind just one case of tabes in which the patient had positive Wassermann findings with symptomatic improvement after intraspinal injections of neosalvarsan by the Swift-Ellis method. He left the hospital, but returned after a year when repeated Wassermann tests corroborated by an outside laboratory failed to give positive tests. The man at his second entry had an advanced case of tabes; he was extremely ataxic and there was no doubt that he was worse, and yet the Wassermann reaction was negative. With reference to intraspinal injection of either salvarsan or salvarsanized serum in paretics, and the following

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remission of symptoms, I think the so-called favorable results are deceptive. We must not delude ourselves into believing that we are causing improvement when the improvement is simply a natural phenomenon incident to the disease. I have seen tabes cases with extreme ataxia come to the hospital with crutches and after a few injections of salvarsan go out without assistance, but even here we must use extreme caution in estimating the beneficial effects. Besides intraspinal injections are not as harmless as one may imagine. In two cases paraplegia followed an injection; in two other cases (not my own) death was the result of intraspinal injection of neosalvarsan. The injection of neosalvarsan, even the small dose of 3 mg., has in many instances caused disastrous effects. Mercurialized serum injections given intraspinally have in my hands caused violent reactions, high temperatures, and extreme pain. In some of the cases there was improvement and in others the reverse. It appears then that at the present time we are not quite prepared to say which is the best mode of treating nervous syphilis. Certain it is that in both tabes and cerebrospinal syphilis the Swift-Ellis treatment, the most harmless of any, deserves still further trial. In paresis we may use this method experimentally, though I am still to see the first case which improved under treatment by any means whatever.

Dr. Mathew A. Reasoner, Washington, D. C.: As to experimental studies of strains of the specific organism of syphilis, a word may not be amiss. As the result of isolating twenty strains from various sources, two from the spinal fluid of patients suffering from nervous syphilis and eighteen from chancres and mucous lesions, we have satisfied ourselves that there are variations in the strains of syphilis we have studied. I am not prepared to say that there are any variations in the morphology of the strains of spirochetes which we have seen. In one strain which I obtained from a man who probably committed suicide three months afterward, I did find choroidal involvement in the rabbit. This same manifestation was observed with our nervous strains. With reference to one statement by Dr. Ball, the serobiologic findings must be considered in conjunction with the others. In the absence of history or manifestations, one positive Wassermann should not be accepted as proof of syphilis. Some one might have mixed the tubes, not to mention other possible sources of error. The test should be repeated. And likewise, in the presence of suspicious manifestations a negative Wassermann will not be accepted as proof that syphilis does not exist. Other methods may be needed to prove it.

Dr. Geo. H. Moody, San Antonio, Tex.: Excluding here a consideration of all syphilitic affections except paresis and tabes, and coming direct to the question of their treatment, we recall that two or three years ago some believed they had had, and were reporting, recoveries as the result of arsenical

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treatment and that a year ago it was practically decided that no cases were getting well. It seems to me that all the papers and discussions at this present meeting lead us definitely to our same former position that nobody is recovering from paresis or from locomotor ataxia. I ask the essayist if they have observed any deleterious results following the administration of arsenic in large doses? I am afraid that in a few of my cases the degeneration in cases of paresis especially, has been more rapid after heroic arsenical administration, such as is being advocated and administered. I am not certain of this and would like to be convinced a little further if there is any evidence leading to such conclusion. Dr. Riggs has reported definitely that there seems to have been marked improvement in many cases. All of us, perhaps, have had similar observations. I know also that many of these patients improved before we gave arsenic at all, under the mercurial treatment, some of them becoming so much improved that for a little while we almost believed them well. The point is; are there any cases in which we are making patients worse as a result of the arsenic. We are now agreed, I think, that formerly we did degenerate many of our cases of paresis and locomotor ataxia by the long administration of heroic doses of the iodids and I believe that none of us are administering them this way to these cases at this time. Shall we later come to believe that the heroic treatment by arsenic may degenerate some of our cases of paresis and tabes?

Dr. Henry F. Stoll, Hartford, Conn.: I would like to ask some one who has given more intraspinal treatments than I, to explain the following: Three patients recently treated were given salvarsan, and within an hour the spinal canal was drained practically dry. The next day the patient was given an intraspinal injection. In this case the Wassermann reaction in the fluid obtained on the first day was ten times as strong as on the second day, i. e., it took ten times as much fluid to get complete inhibition the second day. I do not know whether to interpret this as indicating that the newlv formed spinal fluid had not had time in which to develop fully its Wassermann bodies, or whether it means that some of the salvarsan had gotten into the spinal fluid.

Dr. W. F. Lorenz, Mendota, Wis.: I doubt the value of removing spinal fluid with the hope of increasing the amount of contained arsenic. This practice would seem to me analogous to catheterizing the bladder and by the creation of a vacuum hope to abstract from the blood toxins that may be present. It seems to me that the difference in the chemical composition of cerebrospinal fluid and blood plasma is so great that one must conclude that the former is not a simple transudate or filtrate but the product of a secretory tissue. Viewing it in that light, a negative pressure on one side would hardly affect the composition of the secretion. Several years ago I reported traces of arsenic in the spinal fluid

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after the intravenous administration of salvarsan. In regard to negative Wassermann reactions in syphilitics, I recently succeeded in obtaining a positive reaction with the globulin fraction of such a negative serum. The albumin fraction was negative and antagonistic to the binding forces contained in the globulin. I could by varying the amounts of globulin and albumin from the same serum create a negative or positive reaction.

Dr. James B. Ayer, Boston: I thoroughly believe that we can treat successfully a paretic, only we must get the case early. I believe that the chief value of all this work during the past 5 or 6 years lies here: that it is pushing us back more and more toward the original lesion, so that now the feeling is that we should get patients with nerve syphilis in the secondary stage or the very earliest tertiary stage, and begin the treatment then. It sounds radical, but I believe it is rational. On the other hand, if we do get a patient late, perhaps after he has had syphilis for five years, if we can pick him up on some slight symptom and find that he is suffering from cerebrospinal syphilis, I believe that often the patient can be cured. Perhaps you cannot prove that a seeming neurasthenic is going to be a paretic, but if you find the reactions strongly positive in the blood and spinal fluid, if you get the paretic goldsol reaction, you can feel quite certain that that man is an early paretic. Such patients are being treated successfully at the present time. If in those cases you do not do something, there is every reason to believe that the patient is going to develop general paresis and die from it. Even if you save only one in one thousand, you do more than you would by letting them alone or by giving them a little something to relieve the immediate symptoms. I thoroughly believe in some form of anti-syphilitic serum treatment. I am trying now to get serum into the brain under pressure by the lumbar route. I believe that is a likely form of treatment.

In regard to Dr. Stoll's question of the significance of changes in the spinal fluid on two successive injections, I think the work of my colleague Dr. Solomon on variations in Wassermann and goldsol reactions in different loci of the brain and spinal canal bears on that subject.

Dr. Robert Henry Haskell, Ionia, Mich.: I would like to ask if any of the men using the Ogilvie method of treatment have had herpes zoster developed. After using the Swift-Ellis treatment, two years ago, I treated two cases with Ogilvie's method. Following the initial injection the first patient developed herpes zoster in a rather unusual location. He had been in the hospital for some time and there was no other herpes zoster present. Two months later another case developed herpes zoster following treatment, this time a typical shingles. Was there any etiologic relation between the injection and herpes? Herpes zooster in general paraly-

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sis is not unknown. Arsenical zoster is well recognized in dermatology. Salvarsan intravenously occasionally has herpes following it but search of the literature has not revealed any such complication following salvarsan intraspinally. The French literature contains several instances of cocain administered for spinal anesthesia producing a herpes and following Camp's observations on spinal cords from patients receiving intraspinal salvarsan treatment, the etiologic relation in these cases appears possible and probable.

Dr. Malcolm Sumner Woodbury, Clifton Springs, N. Y.: In regard to the question Dr. Moody has raised, I do not know that I have positive evidence of deleterious results following subdural treatment. I have seen some cases of paresis which I thought were helped by the combined subdural and intravenous methods. We have been using mercurialized salvarsanized serum subdurally supplemented by injections and inunctions of mercury. The patient whose case I will report as bearing on the question, is one I saw during the last few months and who appeared to be simply a psycho-neurotic. There was a lurking suspicion that we might have here a case of very early paresis, though physical signs were absent. To corroberate the diagnosis the usual serobiologic tests were made. As a result we obtained positive Wassermann on both the blood serum and spinal fluid. This patient was put on the ordinary line of treatment intravenously, subdurally and by inunction. At two weeks' intervals he received three doses of the mercurialized salvarsanized serum. He appeared to be feeling generally very much better. Two weeks after the third dose, while coming by train to report for the fourth treatment, he was seized with a sudden manic attack. His case has progressed rapidly since that time to a paretic dementia. Whether the treatment stimulated the onset of the active symptoms I do not know. In regard to the early diagnosis of tabes: it seems to me that it is of considerable value for us to pick out the single, individual symptoms which may appear alone and indicate possible tabes. I would refer to one case, not particularly unusual, in which loss of vibration sense in the legs was the only indication which we could make out on examination, and in which examination of the spinal fluid corroborated the diagnosis.

Dr. George A. Moleen, Denver: Anisocoria, mentioned by Dr. Schaller, is not infrequent where tuberculosis, especially of the deeper set of lymphatic glands, is present, and where they exert an irritative influence from pressure on the cervical sympathetic I have found that to be the case. Such pressure obviously causes dilatation of the pupil on the ipsolateral side. In addition to the symptom mentioned by Dr. Grinker, the early recognition of the loss of the achilles reflex together with loss of the vibratory sense as estimated by the tuning-fork are also important and early symptoms. With reference to paresis: The first patient received five injections of sal-

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varsanized serum, and recovered so thoroughly that he was able to leave the institution and carry on his ordinary occupation, that of salesman, and did well for a year, but took no other treatments and ignored advice so to do, after the injections. But he returned to the hospital with a definite paresis, failed rapidly, and died about a month ago. The second patient had three intraspinal injections of salvarsanized serum followed by mercurialized serum. The mental confusion disappeared following injections, and he then came down with an apoplectiform seizure, which has left its permanent effects, with no mental symptoms. Two other patients, brothers, one with distinct confusion, expansiveness and grandiose ideas, were given intraspinal injections of salvarsanized serum and left the hospital, however, continuing a mercurial course. The brother was definitely paretic, with little mental change, but received the same treatment. These two cases have been rendered stationary for about a year under the treatment. The fifth patient went to California, was given intraspinal treatment and then returned, evidently very much confused and had to be brought home by attendants. He then had about six injections of salvarsanized serum made by the same laboratory in spite of which he progressed rapidly and died about six months after his return. A case of tabes, treated by intraspinal salvarsanized serum and doing well, after involvement of sphincters and marked diminution of visual acuity, was given three salvarsanized serum injections. The patient went to New York where five more injections were given, without changing the cytologic picture or limiting the number of lymphocytes but very little. The use of iodin, particularly in the form of Lugol's solution, alternated with mercury in some form (blue mass by the alimentary tract being my favorite), has to my mind been a means of at least ameliorating or checking the progress in these cases. The method of administering Lugol's solution is five or more drops in water or milk before meals and gradually increasing to fifty or sixty drops.

As to the Wassermann reaction, if we regard it from its fundamental application it is not specific. I favor the clinical findings against the laboratory if there is conflict. The reaction is not specific, but is rather a reaction against lipoid bodies which have been shown to be present in other conditions.

Dr. C. Eugene Riggs, St. Paul: Dr. Ball's paper recalls our own experience with the goldson reaction. In over 90 per cent. of our cases of paresis, we obtained the typical paretic curve, and in over 50 per cent. of our tabetics, we observed a distinctive curve, which we believe to be characteristic of tabes. Also our cerebrospinal luetic cases have almost invariably reacted in the luetic zone. There are some bizarre reactions, however, that we all have noted in this goldsol test. It is not uncommon for us to get the paretic

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curve in a case apparently of pure tabes. I have in mind now one of our cases of tabes, which gave the paretic curve. This patient has been under observation for the past four years. He says he is feeling well. The objective signs still obtain, but the important point is that there has been no development of mental symptoms, no suggestions of paresis. In syphilitic endarteritis one may obtain the paretic curve. A German observer has reported its occurrence in puerperal eclampsia. We have observed it in disseminated sclerosis. We have also obtained this curve in cases of cerebrospinal lues, in which the infection had occurred not more than a year previously. Just what the significance of this reaction is, it will require further observation to definitely determine. Eleven years ago, Dr. Dana called attention to the importance of treatment of the early, "the pre-paretic" stage of paresis. If we can get these patients early there is a possibility of accomplishing definite results. After the disease is well established, and nervous tissues have been disintegrated, they cannot be replaced. Our experience, as well as that of Cotton, Ogilvie and many others, has been that very frequently an amelioration of the disease takes place. Sometimes we are able to make the patient what we call socially possible. He is able to live at home under a restricted environment, much to his own and his family's satisfaction. Even if we obtain only an incomplete remission, the patient may be able to straighten out his business affairs, which usually are in a chaotic condition. We believe it has been demonstrated that frequency of remission is an accomplished fact as a result of either of the intraspinous or of the intensive form of treatment; this means much and of itself makes the treatment of this class of patients well worth while. Answering Dr. Moody's question, we have never seen a patient unfavorably influenced by the use of intraspinous medication. We have under our care at the present time, a paretic who had a remission, and then a relapse. He is now undergoing his second remission. In his case, we have given forty-one intraspinous injections. This man is certainly very greatly improved over what he was when he first consulted us.

Dr. Walter Frank Schaller, San Francisco: In reply to Dr. Grinker I would say that my comparative figures dealt only with the early tabetic cases. As to the tendon reflexes in the lower extremity I have seen all of the cases reported, and practically without exception, have examined all of them for the tendon reflexes. I have been very careful about the technic, preferring the Babinski reflex hammer as I think that this instrument is better than any of the others in common use, and is especially desirable for eliciting the Achilles tendon reflex. Often, instead of striking the patellar tendon with the reflex hammer, I purposely arrest the hammer just before contact in order to eliminate a possible voluntary knee jerk through suggestion. I have often been impressed in

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certain cases by a striking difference between the activity of the Achilles tendon reflex and the patellar tendon reflex; when the former is lost, the latter is found not only to be present but quite lively instead of diminished as one might expect. In regard to Dr. Moleen's remark about the vibration test, I included this test under "Deep Sensibility." We use the Luer fork and very often find the osseous sensibility reduced to 4-5 on the scale.

I was very much interested in what Dr. Ball said about the cell count and I believe that this is one of our most accurate diagnostic tests. I use the Nageotte cell chamber in which one may count cells in a considerable amount of fluid. What Dr. Reasoner and Dr. Pilsbury had to say about the Wassermann reaction appears to me to be deserving of emphasis. I do not accept a Wassermann reaction alone without clinical support as conclusive evidence of syphilis.

Dr. Charles R. Ball, St. Paul: There is one point in my paper which I would like to emphasize particularly: You will recollect that Case 2 was a patient with very active and severe symptoms but negative reactions, except for a mild globulin and lymphocytosis. Case 3, on the other hand, had practically no symptoms, either subjective or objective; and yet his spinal fluid reactions were such as is usually found in paresis. A rational explanation of the difference in the reactions in these two cases may be that in the first case we have an active process with all the congestion and pressure symptoms which accompany active processes but localized and limited in extent. In the second case we have a so-called latent process but widespread in character with a general invasion of the nervous system. In the one case the symptoms are from two sources, first, pressure due to hyperemia and swelling; second, the toxin of the spirochete. Here the symptoms, due to pressure, predominate. In the other case, there is no active process, and consequently, no symptoms which accompany such processes. There is simply a mild and chronic toxemia which in time will have its influence on the nervous elements. To my mind these two cases illustrate the difference between true cerebrospinal syphilis and parasyphilis (tabes and paresis). In the former, we have the symptoms of a localized and limited inflammation of a mechanical nature predominating. The symptoms due to a toxemia are secondary, while in the latter the mechanical or pressure symptoms are secondary in character and the toxic predominate. The rational explanation to me of the cortical atrophy in paresis and the posterior column degeneration in tabes is that it is toxic in origin, in all probability the toxin of the spirochetes.

Dr. Lawrence B. Pilsbury, Lincoln, Neb.: Two years ago I reported twelve or fourteen cases of paresis treated with intravenous injections of salvarsanized serum. Some did badly, two or three at least are still in the hospital appar-

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ently no worse than they were two years ago, and have had absolutely no treatment in the last two years of any sort whatever. Just how much the salvarsanized serum had to do with the remissions I do not know. I also have other cases with prolonged remissions without any treatment. I fear that Dr. Lorenz took too seriously the suggestion that substances might pass over from the blood into the spinal fluid. Yet it does appear from Dr. Barbat's results that a small amount of arsenic appears in the spinal fluid in some cases, and some other investigators have obtained the same result. I believe it is very important that these cases, if they are to be treated intensively, should be treated early. And if the general practitioner would only send at once to the neurologist his cases of "heat and sun stroke," "fainting spells," etc., and let the latter make a thorough examination of the blood and spinal fluid, I think there would be fewer cases of paresis getting to the state hospitals later on in a hopeless stage.

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A NEW TREATMENT FOR PARALYSIS AGITANS

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BOSTON

An Italian author[1] states that in paralysis agitans "no treatment is of any avail," and "there is no authentic case of recovery." A severely distressing malady, of acknowledged incurable nature, and until now of absolutely untreatable form, deserves a few minutes' consideration when even only slight and temporary relief from suffering has been brought to the patient.

The clinical details of the case that determine the diagnosis run as follows:

History.—(M. R.—P. H. No. 3040).—Woman, aged 52, married, German, housewife, first came to the Psychopathic Hospital, June 29, 1915, complaining that the hands trembled, the feet dragged, and the body felt constantly heavy. The trouble began in 1909, when her left arm commenced to tremble. This continued from July to October, and then disappeared. February 2 it began again, and had gradually increased since. The left arm had also grown gradually weaker and stiffer, and its use now amounted to very little. The right arm began to tremble about the same time as the left, but she could use it more. For a year the lower extremities had also showed tremor, and the left one was stiff. Use of the right arm gave an intense shoulder pain. There was much pain in the muscles of the left arm. The tremor ceased with effort, was absent at night, and was worse in excitement. Work was much interrupted by the slowness of the patient's movements, and general stiffness. She was restless and trembled in bed for half an hour before she was able to get to sleep. This all amounted to severe, constant suffering.

The patient had had measles. Menstruation began at 19, hysterectomy had been performed for suspected tumor at 42.

1. Policlinico, Rome, Nov. 21, 1915; abstr., THE JOURNAL A. M. A., Jan. 22, 1916, p. 316.

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This was followed by "pleurisy" and "nervous prostration" Three years after the operation a sponge was removed from the upper abdomen.

The patient's mother died of cholera in 1866 and her father at 62 of pleurisy. Four children were well.

Examintion.—This in general was negative. Below the left breast was the scar of the sponge removal, below the umbilicus the scar of the laparotomy. There was clonic rotation of hands. There were varicose veins on the legs. The tongue extended with tremor. The arm reflexes and patellar jerks were active. The gait was bear-like. The left leg was somewhat spastic; this was more noticeable when walking fast. Oscillations were about four to the second. There was very marked tremor in speech.

A diagnosis of paralysis agitans was made.

Treatment and Results.—The patient was assigned some treatment for the marked tremor in her speech. I was inspired to try *something,* because of the marked improvement obtained in a previous speech case of so-called "essential tremor," in which the patient had been treated for two years without improvement at another hospital. I gave some vowels to be sounded in a prolonged steady fashion, with no intention of attacking the general condition.

To make a long story short, the patient soon returned to relate that the tremor in the hands was a little better; and she felt better. I took the hint, and applied slow motions to the arms and legs to be executed for fifteen minutes, three times a day. The tremor, stiffness and distress gradually grew less. In about two weeks she had an hour of complete relief from all her symptoms — tremor, stiffness, tiredness, pain in arms and heaviness. She could soon go to sleep at once on retiring without tossing about as before for half an hour. Later, she had two hours of complete relief again as above, and at another time went from 7 to 11 o'clock one evening entirely free from symptoms. She was not seen by me at these times, but I have no reason to doubt her words or minimize her claim. She has become so devoted to the exercises that she now takes them merely for the relief they bring. Since February the scales have turned. At that time she had sciatica; and ceased exercising, while confined in bed. With this all the symptoms returned and were as severe as they were before. When seen, June 5, 1916, she had begun to return to her routine again with returning relief.

Let me give the motions in detail:

CONTROL EXERCISES FOR PARALYSIS AGITANS

Patient stands with all clothing constrictions removed. All motions should be executed slowly. The speed is about 3 feet in ten seconds. All sudden, quick or reflex-like motions should be omitted. Each

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exercise is repeated three times. The patient should practice from fifteen to thirty minutes, three times a day.

Exercises:

1. Right arm up to side. Down (shoulder level).
2. Right arm up front. Down.
3. Right arm up back. Down.
4. Right arm flex. Extend.
5. Right hand open. Shut.

6-10. Same for left arm.
11-15. Same for both arms together.

16. Right leg up to side. Down.
17. Right leg up front. Down.
18. Right leg up. Back. Down.
19. Right leg flex. Extend.
20. Toes extend. Flex.

21-25. Same for left leg.

The form of the exercise may be varied from those given, but the important point is the *slowness*.

One case proves little. I should be glad to hear the results from any who try these exercises. A series of cases are now under treatment to be reported later. I find no treatment like this in the books on paralysis agitans.

In one case, at least, the marked temporary relief from severe suffering has amply repaid for all the trouble to do the exercises.

SUMMARY

A marked case of paralysis agitans was treated with slow moving exercises taken for fifteen minutes three times a day, and resulted in (1) a general constant relief of bad feelings, (2) occasionally an hour of complete relief from all distressing symptoms, and (3) a quickly gained respose into sleep on retiring.

The treatment, though giving marked relief from intense suffering even to the point of recovering normal conditions for hours at a time, proves no cure, and the symptoms all returned when the exercises were omitted; but they showed improvement again when resumed, and also again gave relief.

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THE NECESSITY OF REVISING THE NOMENCLATURE OF THE ANATOMY OF THE BRAIN

WILLIAM FULLER, M.D.
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About fifty-five years ago I commenced the study of anatomy by examining the bones by sight, then, by the sense of touch alone, endeavoring to determine to which side of the body each belonged, afterward dissecting the cadaver and learning the names of the parts without reading the text. Later drawings were made from plates found in books, as well as from the subject. In this manner a familiarity with the structure was easily acquired. The names, unusual and frequently those of persons and things without reference to the functions of the parts, required much effort to learn and were difficult to retain in the memory. This difficulty was more apparent when the study of the anatomy of the nervous system was reached, on account of the fact that little was known of the relation of the nerve centers and their connecting tracts, and consequently of their functions.

At this time the dissection of the brain consisted of slicing its substances along certain lines, learning its lobes and fissues, and its ventricles, their boundaries and contents. As for useful information, the soil was barren of practical ideas and was used mostly as a place for the registration of the names of the ancients and of those unfamiliar objects long out of use.

Later the field of knowledge was extended by the use of the microscope, experiments on animals, the tracing of tracts of degeneration, cerebral localization

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and the observations of the parts involved in hemorrages, tumors and other pathologic findings. Other names were added to the list, until at the present time the nomenclature is oppressive and a great hindrance to progress. As it is, the medical student turns in disgust from his futile efforts to conquer a subject which appears to him to be impractical, striving only to learn (not understand) sufficient to pass an examination and to shun the study forever after.

In the study of anatomy, as in all other branches of science, simplicity should be our aim. The use of generalization is a great help to the student. Structure and its supposed function should be considered together in giving a name to any part of the body, so that one reading the history of medicine might be able to compare the progress of one age with that of another. When found necessary, names could be changed to conform with the understanding without much confusion by writing the old names in parenthesis, similar to the change now taking place by the introduction of the decimal system of weight and measures. A gradual change effected in this manner would mark an advance in knowledge at any particular date, and the slight embarrassment suffered would be compensated for by the elimination of a senseless terminology which now oppresses the memory to the exclusion of practical ideas.

I will mention, in illustration, two of the many absurdities of the present nomenclature. The cingulum (girdle), properly named, should be called the superior internal longitudinal commissure, one of the four great longitudinal commissures associating the regions of the memories with the frontal lobe of the brain. This large tract arises in front from the olfactory and marginal convolutions, passes backward over the corpus callosum, then downward and forward and terminates in the hippocampal lobe. A different name is applied to each of three different parts of its course.

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As nearly all parts of the brain encircle the bundles of the internal capsule in the same manner, why select this particular girdle as the cingulum, when they could all be represented together in a single generalization?

Again, there are the "forceps major" and "forceps minor"; I have dissected many brains and have failed to discover them.

Multiplicity of names could be avoided and simplicity attained by the adoption of a systematic generalization of the parts of the central nervous system in connection with that of the whole body. To illustrate, roughly speaking, histologically the body is composed of several segments, modified to conform to the symmetry of the whole. Each segment is composed of all the elements contained in the others, though developments of these elements are unequal. Thus each somatic segment consists of a vertebra, a ventral, or nutritive, arch in front and a dorsal, or neural, arch behind. Superimposed, these segments form cavities, the former enclosing the viscera of nutrition, and the latter the brain and spinal cord. Displacements occur both in the parts of the body and in the viscera of any segment, and both are enervated by the nerve center corresponding to each individual segment, the knowledge of which explains many related reflexes and otherwise obscure symptoms of disease. For example, in pneumogastric irritation the somatic reflexes are manifested on the face. This is true of the central nervous system, except that displacements are limited. Each segment of the nervous system contains the same structural elements, though differently disposed, and often the dissection of one of these throws light on the anatomy of the others.

A comparison of the structure of the cerebrum and cerebellum shows that a hemisphere of each is composed of a hemisphere ganglion, corona radiata, internal capsule, basal ganglia, corpus callosum, commissures of association, ventricles and other parts, all of

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which are distinct and homologous. Why then should the several parts not be designated by the same names and distinguished by an affix, "cerebri" or "cerebelli," as the case might be? A dissection of the ganglia of the segments of the spinal cord displays the same elements in each. A marked difference in the arrangement is that the hemispherical ganglia of the spinal cord are enclosed by the white substance of its several tracts, which becomes everted at the lower part of the medulla oblongata, so that the relation of the gray and white matter is reversed above this locality, in order to accommodate the increase of function required of the cerebellum and cerebrum.

In the description of the anatomy of the spinal cord we are also confronted and confused by the names of the columns of Turck, Rolando, Gowers, Spitzka-Lissaur, Burdach, Goll and Clark. A much more simple and convenient method would be to divide each half of the cord into three columns, and these again into the tracts of which they are composed. Thus, the anterior column, between the anterior median fissure and the motor nerve roots, consists of anterior median and lateral tracts; the lateral column contains the direct cerebral, the direct cerebellar, the crossed pyrimidal and the reticulated tracts; the posterior column is made up of the posterior median and the lateral tracts (Goll and Burdach). Clark's "vesicular column" probably represents the cerebellar element of the spinal segments of the cord, and might be thus designated until a better theory of its function is advanced.

When the American Medical Association met in this city about twenty years ago, I showed in this section castings made from the dissections of the brain. I was asked by several members present of the possibility of repeating them for the purpose of teaching. After having reproduced them, it was found that they were not practical in instructing a large class of students; doubts were also expressed that the nervous

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tracts could be isolated and demonstrated in continuity by dissection, as exhibited in the models.

Photography, however, has dispelled all such doubts, since by the use of the stereoptic camera dissections can be exhibited as seen on the natural subject, specimens of which I am pleased to show you at this time. Lantern slides also can be made to project these pictures on a screen, not flat, but as solid objects. Other methods of instruction are available, even of greater and more permanent advantage to the student.

It affords me great pleasure to do this work, not altogether because of my personal interest in its pursuit, but as well to gratify a desire to do all that I can to assist the student, and also because of the wide scope of its utility in promoting the sciences of psychology, psychiatry, education and eventually justice. Even when we attempt to associate the knowledge acquired of the anatomy and functions of the nervous system with any one of these sciences, we are again in the presence of technical words and phrases of uncertain signification. It appears to me that the science of the mind might be reduced to a few basic principles, easily understood, and from which all the phenomena relating to mind are evolved as the result of reflex action, and that the terminology could be greatly simplified.

ABSTRACT OF DISCUSSION

DR. I. LEON MEYERS, Chicago: The nomenclature of the anatomy of the brain is in a number of instances in great need of revision, as in its present state it is now and then misleading.

The terminology of the cerebellum is obsolete and at variance with the great advances made in the embryology, morphology and physiology of the organ. In dividing the cerebellum into its various lobes, prominence is given to the monticulus—which comprises the culmen and the declive—as a division of the superior vermis. The impression is thus conveyed that the culmen is a part of a unit distinct and separate from the lobulus centralis which is situated anteriorly to it. As a matter of fact, however, the culmen belongs embryologically, morphologically and physiologically to the

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lobulus centralis and these two, together with the lingula, form the lobus anticus (Elliot Smith). The same is true as regards the lobus quadrangularis on the superior surface of the hemisphere. This name would imply that its two parts (the anterior crescentic and posterior crescentic lobules) constitute one unit, whereas in reality the anterior lobule belongs to the lobus anticus, and the posterior lobule to the lobus medius. The prominence given in descriptive anatomy to the sulcus horizontalis magnus—a morphologically unimportant sulcus—has been long ago deprecated by Stroud, and attention is called to this also by Cunningham in his "Text-Book of Anatomy." The name tonsil applied to an important lobule on the inferior surface of the cerebellum in man, originally given to it because it was thought to be an appendage of the uvula, so that both were described under the term of lobus uvulae (Cunningham)—is without justification, as the tonsil is morphologically the anterior part of the alae lobuli medii, or the hemispheres. Elliot Smith has also pointed out the fallacy of dividing the suprapyramidal part of the vermis into a clivus, folium cacuminis and tuber valvulae, all these constituting one common structure. The extent to which the present terminology is misleading is shown by the confusion which resulted when experimental physiologists endeavored to apply the results of their studies on lower animals to the cerebellum of man. We thus find, for example, a man of such eminence as Dr. Rothmann of Berlin identifying the lobulus paramedianus (the tonsil or amygdalus in man) with the gyrus semilunaris inferior; the crus secundum (the lobulus biventer) with the gyrus semilunaris superior; the formatio vermicularis (the lobulus flocculi) with the lobulus biventer; and the crus primum (the lobulus semilunaris superior) with the lobus quadrangularis.

Dr. William Alexander Jones, Minneapolis: Dr. Fuller's efforts to simplify the nomenclature and terminology on the fiber system of the brain should meet with general approval. For many years I taught from some of Dr. Fuller's models and I still study his architecture of the brain with confidence. The illustrations are very clear and they make the brain structure easier to remember.

Dr. Walter B. Swift, Boston: I have taught anatomy of the brain and psychoneuropathology for four years. When I went into this course there were charts and vague models and vague representations of tissue. I soon dispensed with all that, and supplied real brain and tissue sections for study. This has made the teaching much more simple and gives the student a chance to have in mind the visual pictures of the *real tissue* just as it is, not merely visual pictures of charts and models. Dr. Fuller has taken this matter one step farther; and shown not only the tissue and sec-

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tions, but the relations of correlated tracts, and made that subject much easier to learn than from mere flat cross section. I shall use this method more than ever next year.

Dr. D. Booth, St. Louis: The use of proper names in connection with parts of the anatomy is very poor policy, because in that case the student has that abstract fact to remember instead of having the data formulated in his mind. I have always thought it easier to say *iter a tertio ad quartum ventriculum*, than to say aqueduct of Sylvius, because we are dealing with the things which we are trying to demonstrate. Association is a great aid to memory.

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PROGRESSIVE MUSCULAR DYSTROPHY AS AN ENDOCRINE DISEASE *

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In the present paper, the author desires to report a somewhat atypical form of progressive muscular dystrophy, rather resembling Erb's infantile type, of extremely benign and slow progress. In the cases examined it has occurred as a hereditary affection now in the fourth generation. The number of individuals disabled thus far has been fourteen. Of the seven living members with the disease, all but two have been examined by Roentgen ray, and of these five, four show distinct changes in the pineal gland, producing shadows in the roentgenogram. The evidence presented by the cases reported in the present paper, together with that of previous communications in this field, creates a strong probability of the existence of a close relationship between progressive muscular dystrophy and disease of the pineal gland.

In order to understand the evolution that has now placed this disease among the endocrinopathies, a rapid survey of the development of the syndrome, together with the most important contributions thereto in the last few decades, is almost indispensable and is herewith included. The cases studied have been placed at my disposal by Dr. Pearce Bailey, of the Neurological Institute, and for his valuable suggestions and actual assistance, I desire to express my appreciation and my thanks.

Historical.[1]—The first observations on muscular atrophies are found in Boerhaeve's Aphorisms (1709), in which Van Swieten mentions the

* Submitted for publication July 3, 1916.

1. Müller, Albert: Zur Lehre der Dystrophia Muscularis progressiva, Dissert., Kiel, 1902, Surgeon General's Library.

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involvement of the deltoid and that of the intrinsic hand muscles. Later, Charles Bell described syndromes including progressive muscular atrophy. But it was reserved for Aran and Duchenne in 1850 to give the first good description of progressive muscular atrophy. Following them, the French physicians called this type of disease "atrophie musculaire progressive type Duchenne-Aran." Shortly thereafter, Cruveilhier, on the basis of a necropsy, stated that the cause of the disease lay in abnormality of the spinal gray; and ever since then the discussion has waxed hot as to whether the lesion is in the spinal cord or in the muscles themselves. A third theory explained it as a trophoneurosis (Remak, Schneevogt, Jaccoud), with disease of the sympathetic system as a basis. This idea was soon given up. As histological technic improved and positive findings in the spinal cord increased, the supporters of the myelopathic character of the disease seemed to carry the day (Luys, Hayem, Jaffroy and especially Charcot). Charcot, as a result of many investigations, declared the large multipolar ganglion cells of the anterior horns to be the trophic centers of voluntary muscles and that in their alterations the cause of atrophic muscle disease was to be found. Duchenne himself, although originally arrayed on the side of the myopathic nature of the trouble, came around to Charcot's view. So likewise Erb. In spite of the fact that most neuropathologists agreed with Charcot, Friedreich opposed him. In his monograph[2] of 1873 the latter defended the myopathic nature of the trouble in many cases. Thus, he stated that as a result of successive extreme exertions in the same muscle groups, especially in a constitutional diathesis weakening the muscular system's resistance, the atrophies occurred. He described cases following shortly after typhus, typhoid, measles, acute rheumatism, cholera and even after the puerperium, and thereupon declared the condition to be one of "polymyositis chronica progressiva." The changes in the anterior horn cells he considered secondary to the muscle deterioration. But the majority of neurologists stood behind Charcot. Then in 1878 Lichtheim presented a case with high-grade atrophy of great extent in which no cord changes were seen at all, nor were any evident in the peripheral nerves. Therefore, he came to the necessary conclusion that changes in the anterior horn cells are not a sine qua non of the condition, and further, that Charcot's

2. Friedreich, N.: Ueber progressive muskelatrophie; über wahre und falsche Muskelhypertrophie, Berlin, 1873.

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theory could not be the true one. Charcot's preeminence, however, still carried the day. Finally, more recent investigators, after carefully separating all conditions that resembled the progressive muscular atrophies, such as poliomyelitis, chronic multiple neuritis, atrophies with joint lesions, syringomyelia, and so on, from them, two distinct types remained; those that did show spinal cord changes, and those that did not. In 1876 Leyden proposed to separate hereditary forms, on account of their etiology and the localization of the atrophies (pelvic girdle), from the Duchenne-Aran type, and at the same time he showed their resemblance to the hypertrophic type. Lastly, Erb, in 1882, on the basis of a vast variety of cases personally examined by him, declared that there were two distinct types of progressive muscular atrophy, different in their syndrome, localization, evolution, and in their objective behavior, as well as in their anatomic foundation; a spinal atrophic form and a muscular dystrophic form. To this differentiation even the Charcot School assented. But even here it did not take long to discover that there were transitional forms, and in each group of cases intrinsic differences. These depend on the different muscle groups first affected, and on the degree and nature of the muscle changes. Thus have arisen the many confusing types of the disease, and not, as at first thought, distinct diseases. One great work for which we ought to be thankful to Erb for having done for us is that he grouped all these muscular types in one large family, which he called dystrophia muscularis progressiva.[3] In this family he included his juvenile muskelatrophie, pseudohypertrophie infantile form and the hereditary forms. The spinal types he placed under the name "Amyotrophia spinalis progressiva, Duchenne-Aran." Soon thereafter Landouzy and Dejerine (1885) described two cases of "atrophie de l'enfance"[4] in one of which they obtained a necropsy. To their great surprise, they found no changes in the spinal cord, and described the case as a new one, differing entirely from the Duchenne-Aran kind. They called it "myopathie, type facio-scapulo-humeral," and a second type with "intégrité de la face, type scapulo-humeral." Undoubtedly these types are nothing else than the Erb juvenile form, which at times involves the face and again does not.

3. Erb, W.: Juvenile Form d. prog. Muskelatrophie, Deutsch. Arch. f. klin. Med., 1884, xxxiv, 467.

4. Landouzy, L., and Dejerine, J.: De la myopathie atrophique progressive, Rev. de méd., 1885, v, 81.

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In the course of time, all observers, Charcot and Marie included, agreed to accept this classification of Erb's:

1. Dystrophia muscularis progressiva (infantum).
   a. Hypertrophic form
      (1) With pseudohypertrophy
      (2) With true hypertrophy
   b. Atrophic form
      (1) With primary facial involvement (Duchenne's infantile form)
      (2) Without facial involvement. Simple atrophic form.
2. Dystrophia muscularis progressiva juvenum et adultorum. (juvenile form)

It is characteristic of the familial, hereditary forms to exhibit conditions at times of one of these groups, at others, of another group; so that in several generations of the same family individuals will be found to conform with almost any one of the types outlined above. It is so with the family herein studied.

TYPES OF THE DISEASE

Various authors have described atypical forms of muscular dystrophy, that is, they were atypical either in their course or their chronicity, or else in the groups of muscles affected or in the intensity of such impairment; in the presence or absence of contractures; in the slow or rapid involvement of the entire skeletal structure, together with the muscular, making of the patients helpless cripples, and ending in death in a comparatively short time. Some of these important types it may be well here to describe and classify, so that their important characteristics may serve to differentiate them at the same time from the group of cases which I propose to submit further on in this paper. It will also be noticed that as we progress the recent authors show more and more marked a tendency to include symptoms referable to the sympathetic nervous system, including the endocrine glands.

Barsickow's group is late in appearance, slowly progressive and not fatai. It was the first group of great importance, studied by Barsickow[5] in 1871, and consisted of twenty-four cases occurring in two families from one ascendent through five generations. This series resembles most closely the series nerewith to be presented, and yet differs from it in noteworthy particulars. It is presented in extenso for its impor-

5. Barsickow, Hermann: Zwei Familien m. Lipomatosis muscular. progressiva, Dissert., Halle, 1872, Surgeon General's Library, Washington.

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tance. A composite picture of Barsickow's cases showed that the members of the widely scattered families in which they arose were as a rule in good health and lived to be old. The greatgrandfather had only a stiffness in gait and carriage, and yet twenty-three of his descendants (five out of seven of his immediate children) showed muscular disease, not only in function, but also in muscle volume. The members seem to have been attacked without rule, seemingly showing some predisposition in heredity. The children remained healthy if the parents were unaffected, and there were an equal number of males and females attacked. The cause of the original incidence, Barsickow states, was probably lead poisoning, for this original grandfather was a typesetter and had had many attacks of lead colic. In some cases chlorosis, cholera and varioloid acted as preceding agents conducing to the development of the trouble. The onset in seventeen of the cases was at the following ages: five between the ages of 10 and 20, seven between 20 and 30, and five later in life.

It was ushered in by almost simultaneous affection of both an upper and lower limb, or both upper and lower limbs. The serratus anticus was among the first muscles to cause trouble. The most prominent symptom was the disturbed muscular function; in the mildest cases being stiffness and rapid exhaustion, going to absolute paralysis of single muscles, or entire groups in severe cases. The involvement was symmetrical. In most of the cases the diseased muscles gradually disappeared or atrophied, and hypertrophy was very rare. Frequently there was a lordosis with winged scapulae, with protrusion of the shoulders or flattening of the chest. Fibrillary twitching was rare, only once in fact. There was in the skin occasionally loss of pigment or abnormal coloring. Of the eleven who died while under Barsickow's observation, one was over 80 years of age, four over 70 years, three over 60, one 58 and two 33. So that the disease apparently did little to cut short their lives.

Taking up Barsickow's individual cases, those that showed symptoms seemingly not accounted for in the usual syndrome of dystrophia musculorum progressiva, we see in his Case 1 the complaint of rheumatoid pains in many parts of the body, dirty brown pigmented skin and patches of vitiligo. Case 19 was that of a woman of 40 in whom the fingers and toes easily fell asleep and got cold, white and waxy in the winter. Case 7 was that of a woman in whom the disease began at the menopause.

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The reason I mention the above symptoms, which in the light of our present knowledge are referable to a disturbed autonomic nervous system with inclusion of the endocrine glands, is that the theory of Remak, Schneevogt, Jaccoud, and Dumenil relative to the dependence of the affection on such disturbance of the vegetative system has now been shown to have some foundation.

The microscopic examination of the muscle fibers in Barsickow's cases showed changes in the muscle fibrils, some of which were much thickened, while between the primitive muscle bundles were many fat cells and connective tissue. The skin spots appeared in the neighborhood of recently affected muscles. In three cases with necropsy there was no abnormality of the nervous system demonstrable, and yet the author inclined to the idea that the disease is of a "vasomotor-trophic-nerve character." The Barsickow cases undoubtedly belong to the Erb juvenile and adult types.

In Friedreich's group of cases the disease was early in appearance, rapidly progressive, and fatal. Friedreich[6] published the cases of four brothers whose mother was unaffected by the disease, but two of whose maternal uncles died of it in their 15th and 16th years. The brothers cited died at 5, 6, 12 and 16 years of age. Intellectually they were all well advanced. The affection began in the weakness of the lumbar muscles, with constantly increasing difficulty in arising from a sitting posture. Muscular atrophy began in the muscles on each side of the spine at the same time. There then arose a difficulty in raising the arms to the shoulders on both sides. One of the victims died of suffocation from an asthmatic bronchial attack. In this series of cases the rapidity of the course to a fatal termination is the characteristic. The necropsy showed, among other atrophies, that the pectorals were reduced to thin grayish-red, skin-like lamellae, through which the ribs showed.

In Gowers' group the disease was early in appearance, rapid in progress, and was fatal in adolescence. Gowers[7] in a clinical lecture in 1879 reported four brothers, aged 9, 10, 5 and 3 years, all affected, whose parents were both healthy, but whose maternal uncle died at 15 of a wasting muscular disease. These patients had a little difficulty

6. Friedreich, N.: Progress. Muskelatrophie, Berlin, 1873.

7. Gowers, W. R.: Pseudohypertrophic Paralysis, Clinical Lecture, London, 1879.

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in putting their feet down on the floor on account of a tense, shortened Achilles tendon. The extensors of the knee and hip were weak; the flexors of the hip were feeble. The latissimus dorsi and the lower part of the pectorals were gone. There were no sensory changes, the kneejerks were absent and there was a marked lordosis. The upper limbs were weak, although they moved freely. Gowers reports that out of 220 cases that he collected from the literature, 190 of the patients were male and thirty were female. While he mentions Barsickow's cases, yet he excludes them from his statistics, because they were all of the adult type. Some generalizations from these 220 cases are drawn by Gowers: (1) the disease almost never is heard of on the side of the father; (2) the age of onset is an etiologic factor of great importance and occurs in the worst cases before the 6th year, the more severe the case, the earlier it begins; (3) occasionally the disease follows physical injury; (4) shortening and permanent contractions of certain muscles lead to distortions in the positions of the joints; especially contraction of the calf muscles, leading to inability to place the heels on the floor; (5) the patients lose the power of standing at 10 or 12 years of age, and death comes on between 14 and 18 years; (6) of diagnostic importance is not the actual muscular enlargement, but the distribution of the muscular disease, especially the wasting of the lower pectorals and the latissimus dorsi; (7) the later the appearance of the disease, the more slowly it advances; the older the patient, the better the prognosis.

The commentary on these cases is that some of the conclusions, especially the last, are quite contrary to our type of the disease to be described hereinafter.

Erb's[8] types are of the accepted classification of progressive muscular dystrophy. In one of the publications of Erb[8] he gives a general description of the juvenile type of the disease, saying that it begins always before the 20th year and usually with atrophy of the upper arms and shoulder girdle muscles and is often combined with hypertrophy. There are no reactions of degeneration and no muscular fibrillary twitching. The following muscles are almost constantly affected; pectorals (except the clavicular portion), cucullaris, latissimus dorsi, biceps and brachialis anticus and the long supinator. The glutei are

8. Erb, W.: Dystrophia musculorum progressiva, Deutsch. Ztschr. f. Nervenh., 1891, i, 173.

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weak and calves large. The disease lasts from 20 to 40 years with periods of quiescence. He states that this juvenile form is identical with Friedreich's hereditary progressive muscular atrophy. These latter forms belong in the same group with pseudohypertrophy.

In one publication,[8] Erb gives the results of the examination of the available pathologic material of all these forms and says in summarizing that all the noticeable changes are in the muscle fiber chiefly, from an increase in volume to absolute disappearance of the muscle. There is an increase in the muscle nuclei and these are both normally and abnormally placed. There are clefts in the long axis of the muscle between the muscle fibers and an increase of connective tissue is found with a later deposit of fat cells and a thickening of the vessel walls. As a result of the identical pathologic findings in all forms of this elusive muscle disease, he proposes the classification of all these forms as types under the generic term of dystrophia musculorum progressiva. This classification has already been given earlier in this paper.

Among other types the next cases of importance in the development of the semiology of the disease were reported by Prager.[9] Here the patients, two in number, were children of first cousins, who themselves were free of the disease. The importance of the citation of one of these cases is in the fact that while the patient, a woman of 48, had always had a waddling gait and a difficulty in going up-stairs, yet after her first puerperium, which lasted three months, the difficulty in walking was so intensified that she needed support. Her muscular system gradually underwent the usual atrophies in the pectorals, latissimi, trapezius, rhomboids, biceps, deltoids, triceps and supinator longus; but these atrophies were disguised by masses of fat in the arms and legs. To these physical signs, however, were appended the statements: (1) the patient was easily excited and then developed a tremor; (2) there was difficulty in deglutition for fifteen years; (3) dyspnea in cold weather and (4) increase in stools. Further on is the remark that the patient had a "struma" on the neck and complained of urticaria. That is, she was a hyperthyroid subject. We shall find as we go along in the analysis of the symptoms as presented by the author frequent indications of disturbance in the endocrine system. In all probability, had such indications been then understood, there would have resulted a far wider range of symptoms in the muscular dystrophies; for what to the older observers probably appeared negligible in this regard,

9. Prager, M.: Dystrophia musculorum progressiva, Dissert., Erlangen, 1891.

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now assumes great importance. We shall see how more and more often in later years investigators have described conditions present in progressive muscular dystrophy, apart from the actual muscle changes, that are seemingly part of the disease process; which conditions have received but scant reference in the earlier works, presumably because they seemed so utterly adventitious. So Hahn[10] states that skeletal anomalies are often seen and the idea is now that these are part of the condition of progressive muscular dystrophy; and he cites Friedreich that bone atrophy is not secondarily due to disease of the bone through immobility of the joint, but to nervous and trophic influence. Eulenberg[11] had a case which was combined with acromegaly. Hahn's conclusion is that because so many cases show bony changes, there may be some connection between the two.

Bregman[12] cites cases in which together with a "facies myopathique" there was difficulty in looking up and in closing the eyes; inequality of the pupils and of the palpebral fissures. This is interesting from the fact, to be shown from our own cases, of the frequent involvement of the pineal gland; in which condition just such eye muscle difficulties arise (Bailey and Jelliffe[13]). Bregman's cases further showed internal glandular disturbances as follows: In Case 1 the patient could not close the eyes properly and had nyctalopia; also there was intense sweating of the extremities with cyanotic hands and a pulse of 100. In Case 2 there was marked prognathism, the upper teeth standing prominently forward; the skin was mottled like marble. In Case 4 the hands and feet were livid; there was large skeletal development of the hands and feet in contrast to the rest of the skeleton, and marked protrusion of the upper jaw. In Case 5 there was extremely large body growth. In these cases there is manifestly disturbance of both pituitary and thyroid glands.

Cestan and Lejonne[14] publish two cases with contractures and state that it is banal to say that contractures accompany all forms of progres-

10. Hahn, F.: Ueber d. Auftreten v. Contracture bei Dystrophie, Deutsch. Ztschr. f. Nervenh., 1901, xx, 137.

11. Eulenberg: Deutsch. med. Wchnschr., 1896, xxii, 458.

12. Bregman, L. E.: Ein Casuistischer Beitrag z. progressive Muskelatrophie, Deutsch. Ztschr. f. Nervenh., 1899, xiv, Cases 3 and 4.

13. Bailey, Pearce, and Jelliffe, S. E.: Tumors of the Pineal Body, THE ARCHIVES INT. MED., 1911, viii, 851.

14. Cestan and Lejonne: Une myopathie familiale avec rétractions, Nouvelle Iconograph, d. l. Salpétrière, 1902, xv, 38.

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sive muscular dystrophy, but that ordinarily contractures are slight and rarely sufficiently marked to alter the general attitude of the myopathic patient. Schultze[15] presents two cases, both with necropsy, of brother and sister; in the former there was a thinning of the long bones, the humerus being thinner than the middle finger of a normal hand, and the medullary cavity very small. The sister had a stiffness in all the joints of the body, with atrophy of the bones. Schultze states that he had found only two other instances in the literature in which was found bone atrophy with muscular dystrophy, one a case of Friedreich's and the other Le Gendre's.[16] In all these cases the atrophy was a concentric one of the long bones with no diminution in the length. In Le Gentre's case there was also an undue hypertrophy of the genitals, with an enlarged prostate gland in a youth aged 20; while Friedreich's case showed infantilism in the sexual organs, voice and facial expression. The necropsies in Schultze's cases showed no apparent changes in the spinal cord, not even in the ganglion cells, and he therefore concludes that we must look elsewhere than in the nervous system for the cause of this "riddle-like disease," and advises us that we cannot neglect the theory of predisposition, with accidental factors superimposed, such as overstrain, trauma, underfeeding, infection and intoxication.

O. With[17] published a familial type of the disease affecting three boys in one family and sparing the four girls. What interests us in these cases is the involvement of the tonsils, which were hypertrophied, a chronic angina, a hypertrophied lower jaw, with difficulty in mastication and deglutition.

An article of extreme importance on account of the cure of the patient, by Marina,[18] describes a case of a girl of 8½ years of age, who had all the signs of a beginning dystrophy with hypertrophic deltoids and gastrocnemii and so weak in the shoulder girdle group that she could barely lift her arms horizontally. He lost sight of her for five years and then she appeared again entirely well and cured. The

15. Schultze, Fr.: Ueber Combination v. famil. progress. Pseudohypertr. d. muskeln m. Knochenatrophie, Deutsch. Ztschr. f. Nervenh., 1898-1899, xiv.

16. Le Gendre: Gaz. méd. de Paris, 1860, xv, 365.

17. With, Otto: Eine familiare atypische Form d. Dystroph. musc. prog., Dissertation, Freiburg, 1906, Surgeon General's Library.

18. Marina, Alessandro: Gibt es Formes Frustes d. muscularen Dystrophy? Deutsch. med. Wchnschr., 1908, xxxiv, 1087.

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author corresponded personally with Erb in this matter, questioning him as to whether a patient with muscular dystrophy had ever in Erb's experience been cured, or whether he had ever seen "formes frustes" of this disease. Erb replied that he had never seen a single case of "formes frustes"—they had all been progressive—but that he had seen one single case of cure in a young English girl. Erb and Marina agreed that the cure in both of these cases was to be credited to the normal evolution of the patient; presumably to the fact that following the beginning of menstruation, normal development of the internal secretion succeeded, a most pregnant idea in the light of our present knowledge of the interrelation of the ovaries with the other endocrine glands. A fitting companion piece to the above is the case of Levi and Rothschild[19] of a myopathic atrophy which showed considerable improvement on pituitary extract.

Henri Claude[20] in 1908 described a case of familial progressive muscular dystrophy of rather different character from the usual in that the patient had asymmetrical atrophies, more marked on the right side. Besides the muscular condition, the bones on the right side were less developed than on the left side, as shown by roentgenogram, and there were vasomotor changes particularly evident in the right hand, with perspiration and cyanosis. In cold weather the patient could not use this hand on account of the disability thereby occasioned. The temperature of the right hand was constantly lower than the left and the sphygmomanometric pressure was also less on the right side. Claude drew the conclusion from these findings that the whole disease picture does not embrace merely the muscular difficulty, but includes as well the central or peripheral nervous systems.

Jendrassik[21] after citing the cases of Marina and Erb, above mentioned, in which a seemingly spontaneous cure had been accomplished in muscular dystrophy coincident with adolescence, presents two somewhat similar cases, of which the one was a true progressive dystrophy. This patient was a girl who, just before puberty set in, had had the dystrophic process cease, and then after menstruation had been established became entirely cured, even to the return of the knee reflexes.

19. Levi and Rothschild: Rev. neurol., 1907, xv, 613.

20. Claude, Henri: Dystroph. muscul. progressiv. familial, L'encephal, 1908, Pt. 2, p. 512.

21. Jendrassik, Ernst: Gibt es heilbare Fälle von Dystrophia? Deutsch. med. Wchnschr., 1909, xxxvi, 830.

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There had been a precocious body growth between the 10th and 11th years, in which the breasts had participated, growing to a mature size.

Von Werdt[22] describes a case with necropsy, which arose in a woman at the advanced age of 46 and was not familial. It came on after a first pregnancy complicated with phlebitis. She became immediately bed-ridden, and remained so until her death at 63 years. The postmortem examination showed, besides the usual muscle condition, changes in the thyroid, which had developed a large colloid struma with but a little functionating tissue, a small tumor in the spleen, softened adrenals, small pancreas, somewhat atrophic ovaries and small fatty deposits in the liver. This case is interesting and suggestive as having developed after a disturbance of the ovaries due to a late first pregnancy.

Boveri[23] described several members of a family afflicted with muscle dystrophy in which some of the muscles had been entirely replaced by fibrous tissue or fatty tissue, and thought it remarkable that all of the patients had also exophthalmic goiter. Collins and Climenko,[24] among their numerous cases, make mention of the general growth anomalies in several, apart from the general muscular dystrophy. Thus, one patient had a general adenitis with abnormal teeth, especially the incisors, and undescended testicles. Another one had spongy gums, poor and irregularly placed teeth, a high arched palate, long uvula with large tonsils; at the age of 14 this patient was undersized and had as yet no signs of puberty.

Other authors report the presence of anomalies in the skull with asymmetry. Seegard in 1905, on the basis of twenty-one cases, disputed the hereditary factor in the etiology and claimed the entire process to be a metabolic one.

M'Crudden and Sargent,[25] in a careful laboratory study of a case of progressive muscular dystrophy, showed that a condition of hypoglycemia probably underlay the great muscular fatigability. On account of the close relationship between hypoglycemia, muscular

22. Von Werdt, Felix: Ein Fall v. Dystroph. musculor. progressiva m. Sektionsbefund, Zeitschrift f. Pathol., 1908-1909, ii, 577.

23. Boveri, P.: Nevrite hypertroph. familiale, Semaine méd., 1910, xxx, 145.

24. Collins and Climenko: Clinical Study of Fifty Cases of Muscular Dystrophy, Post-Graduate, 25th Anniversary Number, 1908.

25. M'Crudden and Sargent: Hypoglycemia and Progressive Muscular Dystrophy, The Archives Int. Med., 1916, xvii, 465.

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asthenia and deficiency of the adrenals and hypophysis, epinephrin and pituitary extract were administered with resulting improvement in health, strength and weight.

DISCUSSION OF PATHOGENESIS

We see that the more recent investigators have severally and individually been approaching a position regarding progressive muscular dystrophy whose supports are disturbances in the endocrine glands, and have begun to consider the actual muscular disturbances merely as incidents in a widespread affection. And indeed, when we consider the converse of the proposition, namely, that known endocrine glandular disturbances, especially those of the pineal gland, have produced in the muscular, bony, and vasomotor systems conditions very similar to those found in the muscular dystrophies (although not all at one time in any one patient), we must accept some causal relationship between the two. It may be here not amiss to cite, among many observations, some of those that bear directly on this latter proposition. First and foremost is the contribution of Bailey and Jelliffe[18] on tumors of the pineal gland. In their own case, one of the prominent symptoms, and one for which the patient originally sought relief, was difficulty in walking. The several examinations disclosed a hypotonic condition of the muscles of the legs. Furthermore, the patient had grown fat rapidly. In some of the cases appearing in the literature of pineal gland tumors, cited by Bailey and Jelliffe, symptoms and signs are disclosed that are interesting to us now in the light of the findings in progressive muscular dystrophy. We quote some of these. Massot's[26] patient complained that his leg could not support him. Feilchenfeld's[27] patient showed great weakness of the lower extremities with much loss of motor power. One of Nothnagel's[28] patients had a very marked disturbance in gait of a waddling character and the general bodily musculature was weak, and although the man was large and seemingly strongly built, he had much fat deposited throughout. Of interest is Zenner's[29] patient, who developed marked contractures

26. Massot, M.: Note sur un Cas de tumeur cerebrale avec polyuria, Lyon méd., 1872, x, 373.
27. Feilchenfeld, L.: Ein Fall v. Tumor cerebri, Neurol. Centralbl., 1885, iv, 409.
28. Nothnagel, H.: Geschwülst d. Vierhügel, Hydrocephalus-Abfliessen von Cerebralflüssigkeit durch die Nase, Wien. med. Bl., 1888, xi, 162.
29. Zenner: A Case of Tumor of the Pineal Gland, Alienist and Neurol., 1892, xiii, 470.

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in both arms. Von Hoesslin[30] describes a patient with pineal tumor, who, although he had no paralysis, did develop extreme weakness of the lower extremities so that they could no longer support him. Hempel[31] observed a patient who developed general muscular weakness, and later became bedridden on account of contractures in the extremities, so that he was almost unable to move hand or foot. He lay in bed all drawn together. Joukovsky[32] describes an infant of 6 days with pineal cyst who had contractured arms. Marburg's[33] patient developed much fat and showed weakness in one upper extremity and diminished motor power in both lower extremities. Hart[34] published a case in which among the prominent symptoms was increasing muscular fatigability.

Raymond and Claude[35] reported a case most apropos of a glioma of the pineal gland in a boy of 10. He began to grow fat, and grew weak rapidly. Then the left leg became feeble, so that he could scarcely raise it from the ground. He then developed much muscular weakness and finally contractures in the neck and arms arose. Here was a syndrome, exclusive of the other signs of the tumor, strongly suggestive of progressive muscular dystrophy.

Finally, Howell's[36] patient developed extreme weakness in one arm and leg and in the muscles of the back so that he showed a tendency to fall backward. His legs gave way when he tried to walk.

Taking up at this point the manifestations of disease of the pituitary gland, we find it almost banal to refer to the changes in bony structure caused in this way.[37] Especially in hypopituitarism do we see, combined with fatty development and weak musculature, underdevel-

30. Von Hoesslin, R.: Tumor d. Epiphysis Cerebri, München. med. Wchnschr., 1896, xliii, 292.

31. Hempel, K.: Ein Beitrag zur Patholog. d. Glandula Pinealis, Inaug. Dissert., Leipzig, 1901.

32. Joukovsky, V.: Hydrocephalie et tumor d. l. glande pinéale, Rev. mens. d. mal. de l'enf., 1901, xix, 197.

33. Marburg, O.: Zur Kenntniss d. Histologie d. Zirbeldürse, Arb. a. d. Neurol. Institut, 1909, xvii, 217.

34. Hart, C.: Ein Fall v. Angiosarcoma der Glandula pinealis, Berl. klin. Wchnschr., 1909, xvi, 2298.

35. Raymond and Claude: Les tumeurs de la glande pinéale, Bull. de l'Acad. de méd., Paris, 1910, lxiii, 265.

36. Howell: Tumors of the Pineal Body, Proc. Roy. Soc. Med., 1910, iii, 77.

37. Cushing, H.: The Pituitary Body and Its Disorders, 1912. Timme, W.: Pituitary Disease, New York Med. Jour., 1915, cii, 801.

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oped bony tissue[38] with rarefaction. Insufficiency of the suprarenals produces great fatigability in the muscular system;[39] thyroid insufficiency, through its effects upon metabolism, produces occasionally changes in joints leading to their immobility and to contractures in the muscles and tendons controlling them.

By combinations of these disturbances many of the symptoms exhibited in the cases of progressive muscular dystrophy might be produced. It is highly suggestive that the variations in this most elusive disease, which are enlarged on by almost every investigator, have given rise to almost as many types or forms of the disease as there are investigators. And this is not to be wondered at if the syndrome depends on as many variables as are represented by the endocrine glands.

The findings at necropsy in the various types of progressive muscular dystrophy do not contradict an internal glandular basis for the disease. Kollarits[40] quotes Blocqu and Marinesco,[41] Flandre,[42] and Pennato[43] to show that a relatively negative result in so far as the nervous system is concerned, was obtained by the more recent investigators. This is to say, in by far the greater number of cases that went to necropsy, no important changes were found in the cerebrospinal system, although in a small number there were minor changes in the anterior horn cells of an atrophic nature. These minor changes are not always the same in the different cases and are not proportionate to the clinical disturbances. "For this reason (quoting Kollarits) Erb stated that the dystrophy is probably dependent on functional disturbances of trophic centers not demonstrable anatomically." The actual muscle changes found histologically agree in the main with one another in the different investigations (Spiller.[44] Münzer,[45] Fulda,[46]

38. Timme, W.: A case of Polyglandular Insufficiency with Akromikria, Boston Med. and Surg. Jour., 1915, clxxii, 828; Conferences of New York Neurological Institute.

39. Cannon, W.: Bodily Changes in Pain, Fear, Hunger and Rage, D. Appleton & Co., 1915.

40. Kollarits, Jeno: Beitrag zur Kenntniss d. anat. Grundlage d. Muskelatrophie, Budapest Clinic, Division 2, Deutsch. Arch. f. klin. Med., 1901, lxx, 157.

41. Blocque and Marinesco: Arch. d. Neurol., 1893, xxv, 189.

42. Flandre: These de Paris, 1893.

43. Pennato: Clin. med. ital., 1898, xxxvii, 31.

44. Spiller, W. G.: New York Med. Rec., 1898, liv, 9.

45. Münzer: Ztschr. f. klin. Med., 1893, xxii, 564.

46. Fulda: Deutsch. Arch. f. klin. Med., 1894-1895, liv, 525.

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Kollarits[40]). The muscles show a large amount of fatty tissue between the muscle fibers, and a few of the muscle fibers with rounded ends. The width of the muscle fiber varies from the normal and the muscle nuclei are increased in number and are grouped together. There is splitting and vacuolization of the muscle fibers and entire disappearance of certain muscles. At times the entire muscle seems to be a gray, fatty mass (Schaedel[47]). The bony changes are at times atrophic in character and at times hypertrophic (Eulenberg[11]).

We can certainly say that postmortem evidence points to a metabolic disturbance. But metabolism in the body is so closely interwoven with the activities of the internal glandular system and the vegetative nervous system that they can hardly be considered apart.

AUTHOR'S TYPE OF FAMILIAL PROGRESSIVE MUSCULAR DYSTROPHY

It has been my good fortune to observe several members of a large family during the past year who were affected with a type of progressive muscular dystrophy rather resembling the classic Erb's juvenile form, but differing from it in some particulars. The family tree is herewith given (Fig. 11). The onset and progress of the disease are graphically described by one of those afflicted, a highly intelligent graduate student of one of our foremost universities, and I cannot do better than to give his own description of this insidious affection:

The muscular disease which afflicts our family is similar in all the cases I have investigated. In my own case the trouble appeared at the age of 3, and was noticed by my peculiar method of arising from the floor. The same difficulty was observed in my brother and my sister during early childhood. A general lack of development of the muscles throughout the body marks our disease; and there is an accompanying tightening of some of the tendons which limits movement. In the case of my brother the heels are raised from the floor as much as three inches when he stands erect; and my sister is compelled to walk on her toes and the front of her foot so that a special shoe is needed. With the advance of years the difficulty in arising becomes more pronounced. I find it much harder to get up from the floor now than it was seven or eight years ago. My sister, father, uncle and aunt, who have the trouble, cannot arise if they fall down, and they cannot get up from a chair of ordinary height. My sister has put on much weight lately, resembling in this my grandfather, who also had the disease. The latter could not roll over in bed, arise, or walk alone. The difficulty in climbing stairs, and in general, in all movement becomes more pronounced with advancing years. The contractions with the drawing-up feature become worse during the growth from childhood to maturity. There is some

47. Schaedel, Wilhelm: Zur Lehre v. d. Dystrophia musculorum progressiva, Dissert., Kiel, 1909.

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Fig. 1.—Attitude at rest, standing, showing the contractions at the elbow and phalangeal joints; the disparity between fairly normal calves and atrophied thighs; also the leaning forward of the head and the retraction of the chest.

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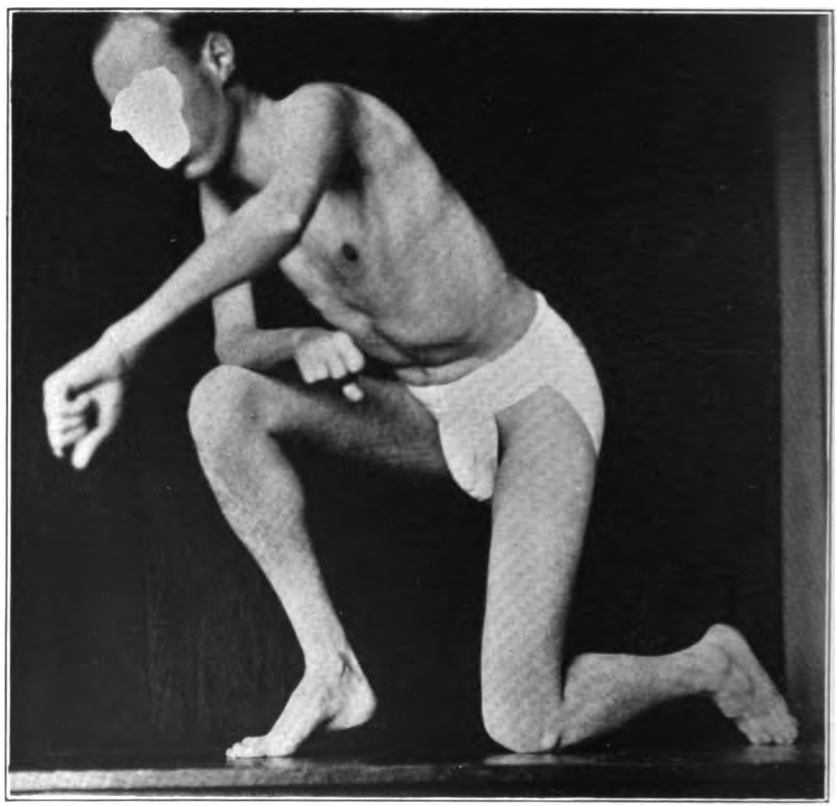

Fig. 2.—Manner of arising from the floor; climbing up on thighs.

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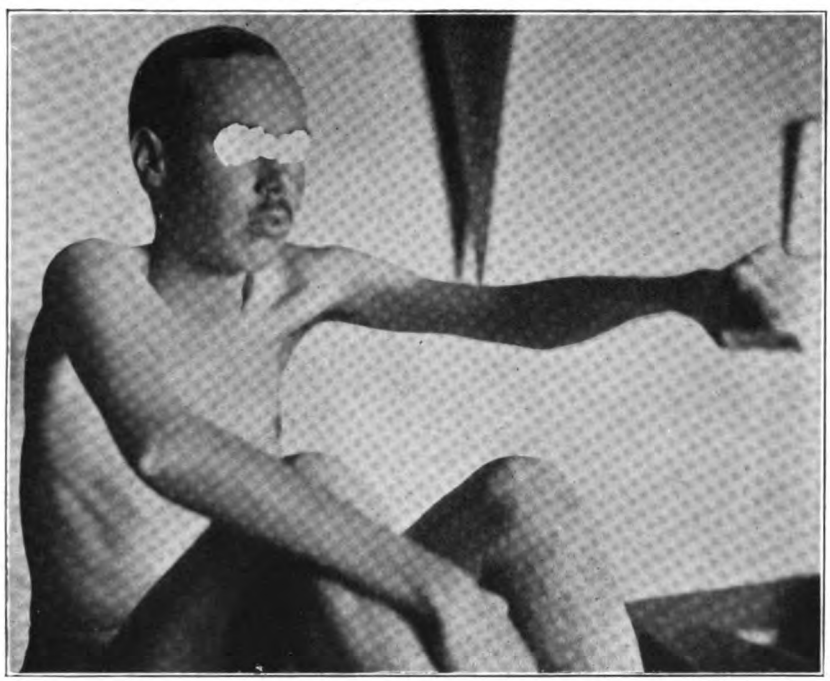

Fig. 3.—The isolated muscle bundle that still remains of the left pectoralis, the rest of the muscle being gone; note the funnel-like neck sunken into the aperture made by the clavicles, and the atrophic arm muscle.

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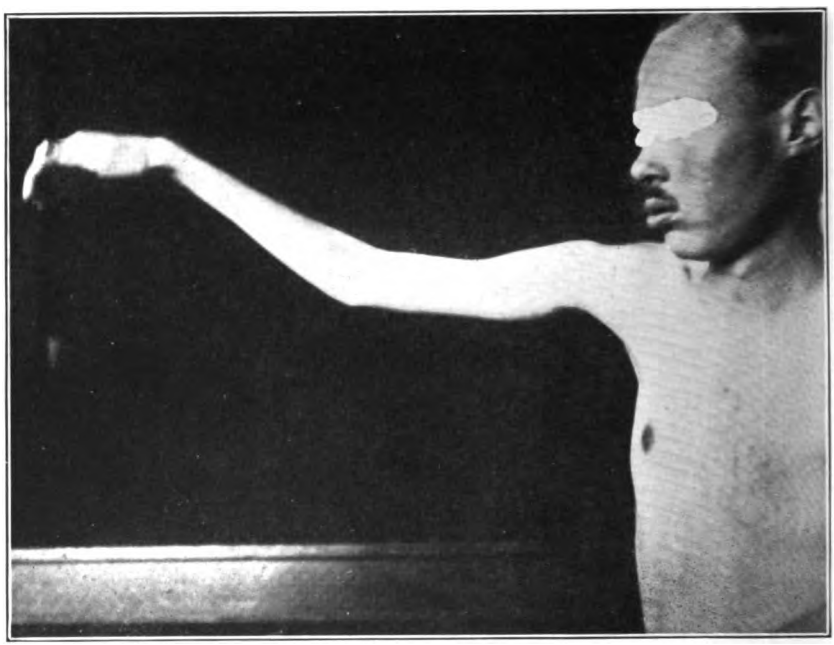

Fig. 4.—Contractions in the tendons of the arm, the picture giving the extreme limit of extension of the arms and hands possible. Note also the pouting of the lips with a suggestion of the "facies myopathique."

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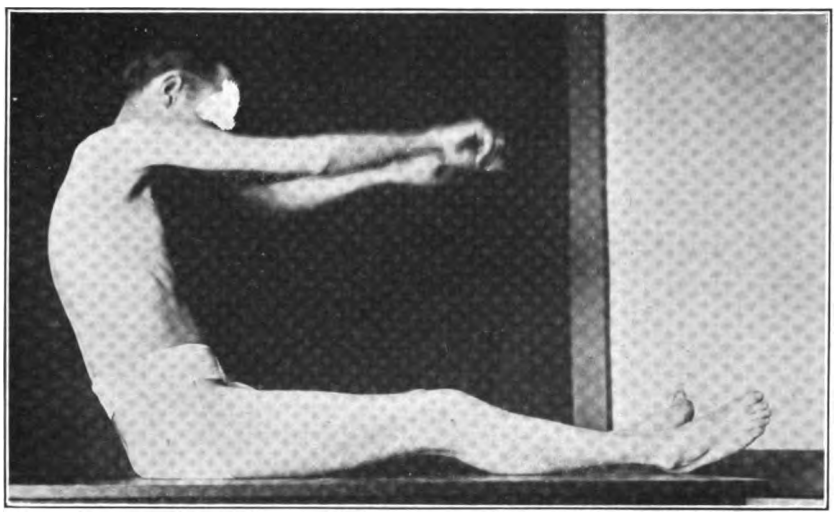

Fig. 5.—Extreme extent of flexion of trunk on thighs that is possible, with legs extended fully, this deficiency being due to the short tendons of the thigh and leg.

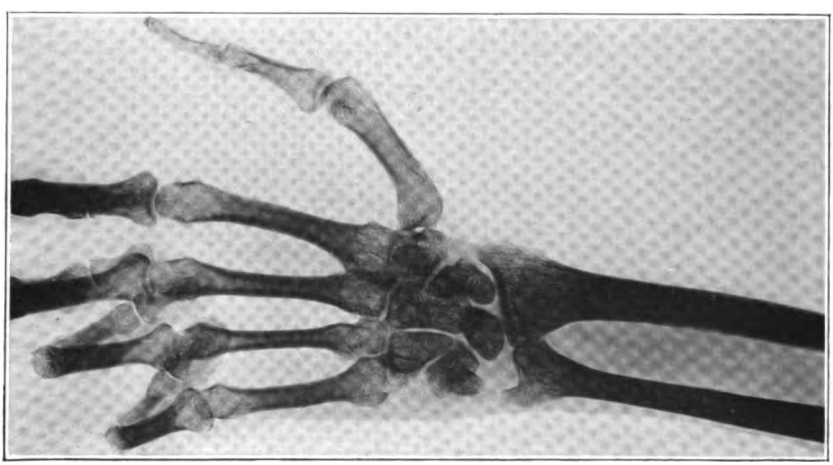

Fig. 6.—Roentgenogram of the forearm and hand, showing the thinness of both ulna and radius and of the phalanges (Patient 24, Fig. 11).

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Patient 14, Fig. 11).

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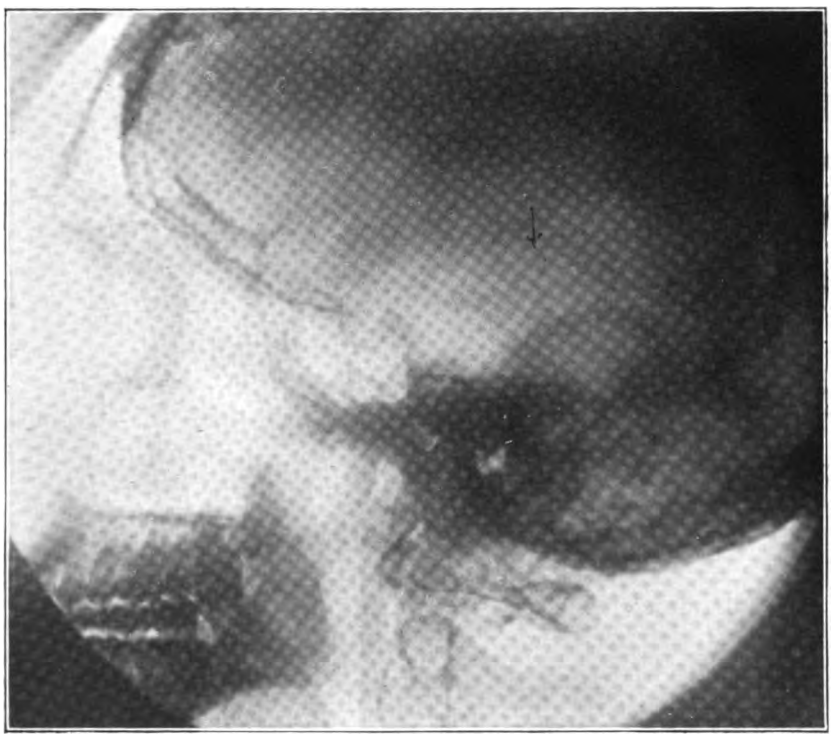

Fig. 8.—Roentgenogram of skull of Patient 26 (Fig. 11), a youth 21 years of age, showing shadow in the pineal region.

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Fig. 9.—Roentgenogram of skull of Patient 27 (Fig. 11), a girl 19 years of age. Here the shadow is not so distinct as in the others, but still is present.

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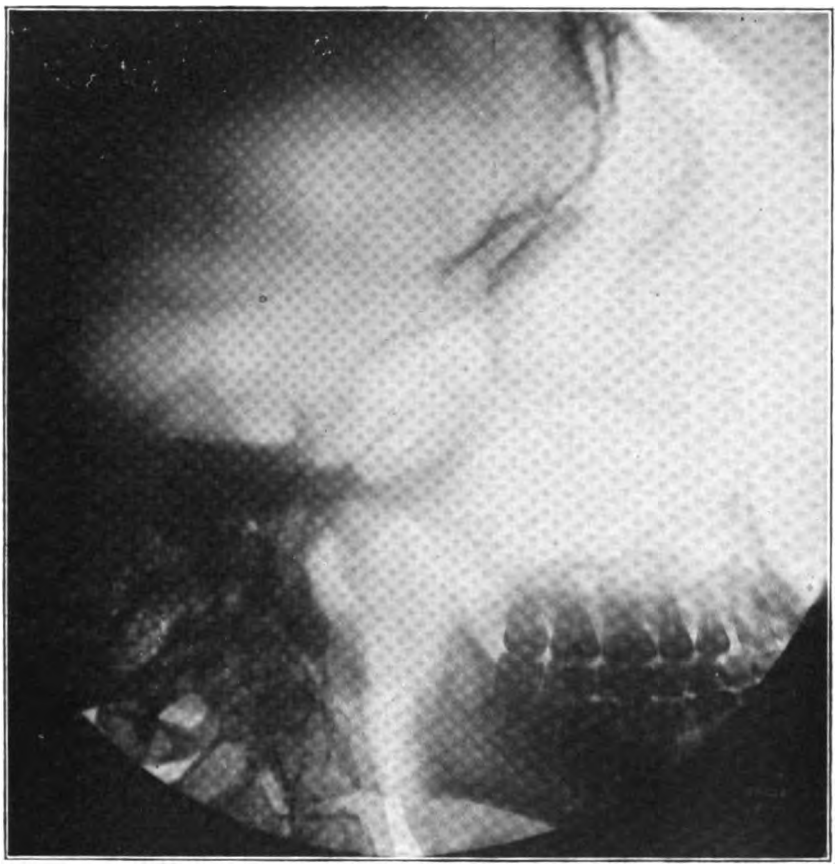

Fig. 10.—Roentgenogram of skull of patient shown in Figures from 1 to 5, inclusive. The shadows in the pineal region are marked (Patient 24, Fig. 11).

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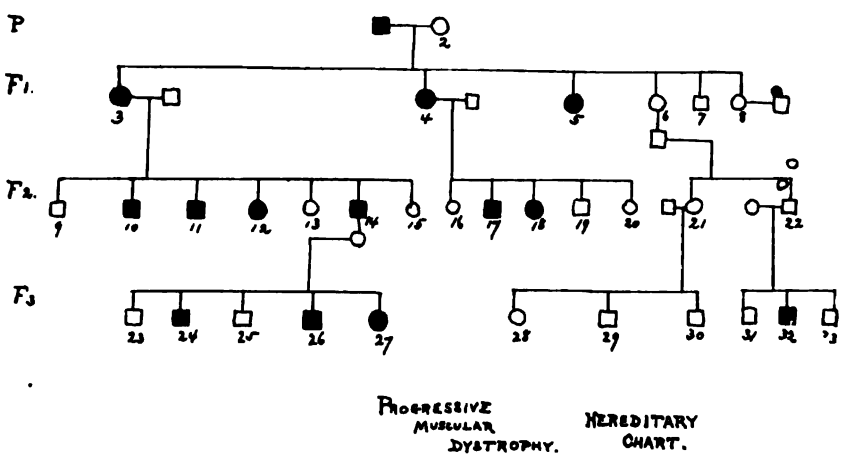

Fig. 11.—Heredity chart of the family in which the dystrophy cases occurred, those in black being the individuals affected.

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reason to believe that this contraction continues slowly through the periods of maturity and especially of senescence. The general health of the affected individuals is very good. I cannot remember of illness, or of the report of illness in the case of my father or my uncle; the family is long lived and intellectually sound. There is no bodily pain connected with the disease. There is only a great physical limitation with its accompanying embarrassment. Tradition has it that the original case of the disease arose at the age of 12 following an injury in the field.

Of the fourteen members of the family through three generations who were affected, six have died, at the ages of 88, 73, 71, 69, 64, and 53. Of those living, three are 69, 66, and 64 years old, while the rest are of the present generation in the teens or twenties. That is, the span of life seems rather to be above the average. In this regard, the affection differs from most of those heretofore cited. Gowers,[5] out of a study of 220 cases that he collected, declared that the earlier the disease begins the more severe it is, its usual onset being before 6. Out of thirty of his own patients, twenty-four died between 10 and 20; and the oldest lived to be only 40. He also gave the predominant male to female ratio of 190 to 30. In our cases, nine were male and five female. Barsickow,[5] in his study of twenty-four cases, while giving an age at death closely approximating ours, yet declared the onset in all of seventeen known cases to be after the 10th year and mostly between the 20th and 30th years, truly an adult type. So that our cases differ from these two types in the combination of an early onset and a long, rather benign course.

Symptomatology: We have been able either personally or through their own physicians to examine thus far six of the seven living members of the family that have the disease. Patient 24 (Fig. 11) was the one originally examined at the Neurological Institute by us. His examination showed the following:

The patient is a man of 23 years, a student. The station is good, though the attitude is somewhat faulty, as is seen by the photograph (Fig. 1). His head leans forward, with chest retreating; there is some lordosis, and the arms are held at the side partially contracted at the elbow and wrist joints. The gait is peculiar in that he stoops his shoulders forwards and toes out. If he doesn't stoop his shoulders, his heels will not touch the floor. There is little or no spring in his walk, his whole body jerking with each foot fall. His weight when he is stripped is 108 pounds.

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All the deep and superficial reflexes are normal and equal on both sides; there is no Babinski response and there is no clonus.

There are no sensory changes objectively to be elicited, excepting some pain on pressure over the fourth dorsal vertebra; subjectively he complains of no pain whatever excepting at times "rheumatic" pains in the joints. This soreness in the joints all the affected members of the family experience. Those unaffected with the dystrophy do not have the joint pains.

There are no signs of involvement of any of the cranial nerves, with exception possibly of those controlling the eye muscles. Every dystrophic member of the family gives a history of eye trouble and must wear glasses. These ocular muscle disturbances are important as referring possibly to the pineal gland in its relation to the anterior corpora quadrigemina. His father, also affected with dystrophy, awoke suddenly one morning, blind, and never entirely recovered his vision. There is no difficulty in swallowing or in speech, and the tongue is protruded in the middle line.

The only external signs of an involvement of the internal glandular system is in the spacing of the incisors, and in the prognathism of the upper jaw. We shall later have occasion to point out the extreme probability of the dependence of the entire syndrome, the bony atrophy, fatty deposits in the muscles, etc., on such a disturbance of the endocrine system.

There is no arteriosclerosis; the pulse rate varies from 68 to 72, increasing on slight muscular effort to 90. The blood pressure, systolic, varies from 125 to 145; diastolic, from 75 to 100.

The patient is highly intelligent, a postgraduate student at an important university, fitting himself for the philosophical degree. His auditory memory is good, while his visual memory is very poor. He is fairly frequently depressed, seclusive and shut-in and has a reaction of embarrassment in company. He feels his disability keenly.

Urine and blood are quite normal in all particulars. The Wassermann reaction in the serum is negative.

It is in the muscular system that the chief characteristics of the condition lie. The first striking feature is the almost entire absence of the pectorals of both sides, with the exception of the clavicular strands and a few strands of the pectoralis minor (Fig. 3). Both deltoids are very atrophic, and the intrinsic neck muscles are so much

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so that the neck seems to fit like a funnel into the aperture made by the clavicles and sternum instead of being attached thereto. The trapezius, the serratus magnus, the rhomboids are only partially atrophied. The biceps and triceps are much atrophied; the long supinators less so, while the muscles of the forearm are only slightly affected. In the leg the quadriceps and the adductors are smaller than normal and quite inefficient, so that walking up stairs or getting up from a squatting position is a task. It is accomplished by climbing up on the thighs by means of the arms (Fig. 2). Of the hypertrophies that are present, the infraspinatus is moderately enlarged, as are the calf muscles. The facial muscles show a slight atrophy, with everted and pouting lips, although there is no difficulty in whistling. The electrical excitability is somewhat increased to faradism in all the muscles, while the galvanic response is unaltered in degree and quality, that is, there is no reaction of degeneration, no myoclonus, or myoidema; there is no asynergy and no incoordination. There are no actual paralyses, but simply weaknesses. The patient can hold his arms out from his body at the level of the shoulder for one and one-half minutes without fatigue. There then ensues a tremor of the entire limb, and he must let the arms fall. The various measurements of the circumference of the limbs are herewith given in millimeters:

		Right	Left
Upper arm.....	Upper	230	225
	Middle	190	190
	Elbow	193	190
Forearm	Largest	200	202
	Smallest	125	127
Thigh	Upper	465	465
	Middle	420	415
	Knee	320	330
Lower leg.....	Calf	335	325
	Thinnest	173	175

There are contractions in the tendons of the arm muscles and of the calf muscles on both sides—contractions, and not contractures. That is to say, by assuming such positions as will give little tension to the muscles of the particular member examined, the patient recognizes no difficulty in fully extending that limb. Thus, if he relaxes the calf muscles by stooping forward in walking, his heels touch the ground; but if he stands fully erect with shoulders thrown back, then his heels

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do not touch the ground on account of the short Achilles tendons. The photograph showing the arm outstretched to its fullest extent, gives an idea of the shortening of the arm flexor tendons, in the resulting flexion of the fingers and wrist (Fig. 4). When the wrist is fully flexed, the fingers can be extended; when the fingers are fully extended and kept in that position, the wrist cannot be extended at the same time. It is these contractions that give the patient his greatest difficulty, for they impede the free use of his limbs and are progressively, although slowly, increasing (Fig. 5).

The Roentgen ray shows that the bones of the arm and forearm are very small and thin, though not shorter than the normal (Fig. 6). The skull roentgenogram shows a normal sella turcica, but there are several shadows in the region of the pineal gland (Fig. 10). These shadows are three in number, and although discrete, range closely together.

Three other members of this immediate family, Patient 14 (Fig. 11), father of this man, and Patients 26 and 27, brother and sister of the patient, give similar physical signs and symptoms to those exhibited by this patient, with some minor differences. Thus, his sister, 19 years of age, is quite adipose, and has more marked tendon contractions than the others. She constantly walks on her toes as a result of the Achilles tendon shortening (pes equinus), and must have special shoes to accommodate this malposition of the foot to her gait. The brother, aged 21, is in many respects the counterpart of the patient, only not quite so far advanced. The father, aged 65, cannot get up from a sitting position without help. There is the same difficulty in all of these members of the family in going up stairs, almost an impossibility to lift themselves from step to step. But all of them are active mentally. The father still attends to his duties as a minister. The one point that they all have in common, and this we wish to emphasize most strongly, is that the roentgenogram in each instance shows shadows in the region of the pineal gland. In the sister's case these shadows are very faint, but still present (Figs. 7, 8 and 9).

One member, No. 32, of a collateral branch of this family, but still descended from the common ancestor No. 1, is affected by a type of the disease which is sufficiently distinct from that of the others to merit individual description. He is a schoolboy of 15, living in a small Pennsylvania town. His difficulty seemingly followed an attack of

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chickenpox four years ago. He has no difficulty in squatting down or in arising from the floor, thereby differing from every other member with the disease. Within six months of the onset of the disease, his joints became stiff and the seat of calcareous deposits. Various tendon contractions followed, notably the Achilles and the flexors of the fingers. Muscular atrophies, especially of the deltoids, pectorals, trapezius, biceps, and sternocleidomastoids succeeded. The pectorals are very deficient, the sternocleido muscles also, almost like a lead-pencil in thickness. The entire bodily musculature seems to be deficient in volume and gives the appearance of a big bony frame with insufficient covering. The myotatic irritability is increased in the biceps and deltoids. The bony framework is large, the middle finger measuring 105 mm. from the metacarpophalangeal joint to the tip. Both ankles are deformed, with a marked protrusion of the tarsals inwards and a pes planus. The foot cannot be flexed dorsally on account of a short Achilles tendon, or ventrally on account of a short tibialis anticus. This gives the patient a rather queer shuffling gait. There are calcareous nodules on many of the phalanges, especially at the terminal joints. On account of atrophic supinators, supination can be carried out only to a very limited extent by the biceps assisting. This syndrome is rather different from that of the other members, in that it began at the comparatively late age of 11 and that it was not accompanied by difficulty in arising or in going up stairs and in its marked disposition to affect the bony skeleton. The Roentgen ray also in this case did not show the pineal gland shadows, but did show a large sella turcica. As the boy is but 15, it is within the realm of probability that within a few years the shadows will appear.

As the members of the various branches of the family are widely scattered, indeed from coast to coast, I have not personally been able as yet to examine all those affected with the disease. The roentgenograms were made by different roentgenologists in various parts of the country, including Dr. Leix of Los Angeles, Dr. Bowen of Philadelphia and Dr. Evans of New York.

To recapitulate, then, we have a somewhat atypical form of progressive muscular dystrophy, rather resembling Erb's infantile type, of extremely benign and slow progress. It occurs as a hereditary affection, now in the fourth generation, which has disabled fourteen individuals. Of the seven living members with the disease, all but two

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have been examined thus far by Roentgen ray, and of these five, four show distinct changes in the pineal gland producing shadows in the roentgenogram. The fifth, a youth of 15 years, whose affliction is rather of different type than the others, in that the bony growth is abnormal, shows an enlarged sella turcica, but no shadows in the pineal as yet. Weighing all the evidence that is advanced by previous investigators to show derangement of the internal glandular balance in cases of progressive muscular dystrophy, and giving due significance especially to the changes produced by tumors and diseases of the pineal gland on the various tissues of the body, changes that resemble in character, if not entirely in degree or disposition those present in progressive muscular dystrophy, we must admit the extreme probability of a causal relationship between the two. If to this probability we actually adduce the evidence shown by cases of progressive muscular dystrophy of changes in the pineal gland demonstrated by roentgenology, the probability approaches pretty closely to proof that disturbances of the pineal gland play an important rôle in the pathogeny of progressive muscular dystrophy.

In criticism of this deduction, there may be advanced the statement that frequently otherwise normal individuals show shadows in the pineal gland. In answer thereto, Schüller of Vienna, who was the first to demonstrate the identity of the shadows with deposits in the pineal gland, states that in older persons who already have reached the stage of calcification of the arteries, the shadows are but evidence of such calcified bodies in the pineal gland. But here we have three of the four patients under 24 years of age. The author personally examined about 150 roentgenograms of the skull in the laboratory of Dr. Lewis G. Cole of New York, and found only about 2 per cent. with pineal shadow.

To ask why changes in the ovaries or thyroid or pituitary gland, all of which changes have had their part in the production of symptoms in many of the cases of progressive muscular dystrophy herein cited, should be laid at the door of a diseased pineal gland, brings up the whole question of the interdependence of the endocrine glands. Suffice it to say that the pineal gland is supposed to cease its function at puberty. If earlier or later, then the other glands partially compensate, producing their own particular symptoms in the endeavor to right the wrong. Occasionally this wrong is righted and a cure of

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the condition produced by the perverted pineal gland results. Thus have come about the several cures reported by Erb, Marina, Levi and Rothschild, and Jendrassik. In each of these cases a rapidly developing gonadal system incidental to puberty produced the cure.

A final report of all the members of this family will be presented by me within the coming year, together with a short disquisition on the therapy of the condition based on the foregoing theory of the pathogenesis of progressive muscular dystrophy, namely, that the disease must be classed as an endocrinopathy.

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ABSTRACT OF DISCUSSION

DR. A. L. SKOOG, Kansas City: This subject should be investigated much more thoroughly than it has been. Not only the pineal gland, but probably all the endocrine glands should be included in these investigations in cases that might come to necropsy. Last July a boy, 9 years of age, had his first evidences of hyper-pinealism in a rapid, precocious development, with enormous appetite, polyuria, and an extremely rapid development of all the primary and secondary sexual characteristics. He grew rapidly for a few months and quickly developed symptoms of gradually increasing intracranial pressure. In the terminal stage he began to emaciate, became bedridden, and had contractures, the arms and legs being contracted and held extremely rigid. He declined mentally after a short time and remained in that state for about four or five months and died last February. A partial necropsy was granted, only the brain being promised for my possession. We found a marked internal hydrocephalus, sufficient to cause quite a bulging just posterior to the chiasm, and a tumor of the pineal gland about the size of a large hen's egg. On section of this tumor I found none of the sand bodies so frequently found in the pineal gland. The tumor was of the adenoma type, with multiple large cells, in fact the cells were larger and apparently more succulent than normal pineal gland cells. The Roentgen ray in this case did not reveal anything, except that the gradual increase of intracranial pressure had changed the cranial bones. The hypophysis was practically normal. I never did believe that he had hypophyseal disease, which was suspected by other examiners. I would like to ask Dr. Timme if he has any explanation for the varying symptomatology which we have had reported from pineal gland disturbances, and if there has as yet been determined any syndrome for hypo- and hyper-pinealism.

DR. M. A. BLISS, St. Louis: What form of glandular therapy has Dr. Timme used in his cases?

DR. LEWIS JOHN POLLOCK, Chicago: I did not quite understand the age of the patient, the girl in whom it was seen that there were quite distinct markings of convolutions on the skull. As I understand, she had marked adiposity. I would ask Drs. Timme and Bailey if they correlated these findings with changes in the pineal gland, or a condition which occurred as the result of a secondary hydrocephalus with a cystic third ventricle and pressure on the pituitary body, which many cases of pineal gland tumor show.

DR. GEORGE A. MOLEEN, Denver: As soon as I get back to Denver I am going to have the heads of three brothers roentgenographed. The first of these three cases came under my observation some six or seven years ago, following the recovery of incipient dystrophy by the use of thyroid substance. I placed this patient on that treatment, since which

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time I believe there has been but little if any change in his condition. Three years afterwards I chanced to see the other two brothers and they were typical cases. The profound weakness was very striking. When I say that in getting up from the recumbent position one of them had to support his head before he could even start the movement, the weakness of the scaleni muscles is apparent. One of my cases, who presented a forearm atrophy with profound weakness of both hands, and who had a registration with a dynamometer of 120, came up to 280 in about four months after the use of small doses of thyroid and later resumed his occupation as railroad fireman.

Dr. Albert E. Sterne, Indianapolis: A case I do not in any sense correlate to the findings which Dr. Timme has made, as the roentgenograms of the head have as yet shown no sure marks of any lesion in the pineal gland, is one involving three brothers, one of about 11, one about 8, and the youngest about 6, the older showing a very marked dystrophy, the second a slightly lesser grade, and the youngest still less, all progressive. In all three cases Dr. Moon found the streptococcus viridans in the blood. Despite this finding I did not regard the streptococcus viridans as having any relationship whatever, as far as I could determine, to the presence of this dystrophy. But it does seem more than passing strange that one should find a practically virulent coccus in the blood of all three brothers. To be sure one might say, on the other hand, that the focus from which the infection in the blood came might have existed independently in the three children, or, perhaps, one infected the others, but so far as we could determine there were no apparent foci in the tonsils or around the teeth through which we could find that any infection occurred. We might devote more attention to the blood in these cases than we have heretofore done.

Dr. Walter Timme, New York: As to the various symptoms in dystrophy, this is a difficult question to answer because before one notices the signs relating to the dystrophy, produced, as a rule, in pineal gland tumor, you get the general signs of the tumor itself, and as soon as you get these signs the other signs become either adventitious or may be based on the actual condition of internal hydrocephalus which you get as a result. But if that tumor does not grow very rapidly, as occasionally occurs, then we do get pineal gland involvement that shows these symptoms: First, difficulty in the use of the ocular muscles due to the fact that the pineal gland impinges on the anterior corpora quadrigemina. And there have been ocular disturbances in every one of our cases that had the disease, and no ocular disturbance in those that did not have the disease. Secondly in pineal gland disease there are symptoms referable to disturbance of the sex glands. Normally, the pineal gland is supposed to have ceased its function at puberty, but if it does not cease its function at that time we

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may get infantilism, or sometimes even sexual reversion, a reversion in type of the distribution in the pubic hair, undescended testicles, etc. In a hypo-pineal condition we do get precocious puberty and all the conditions that go with that finding. The dystrophy symptoms that come with pineal gland affection are those due to hypo-pinealism, as a rule, that is, in which the pineal gland is destroyed too early, when you do not get sex anomalies, but growth anomalies. Most cases will appear like old men even in their youth. They give the appearance of a large bony framework with too little muscle to cover it. Dr. Graves asked about the glandular therapy applying in these cases. You can not apply glandular therapy in these cases, or in any cases, according to a fixed rule. You do not treat the disease, but the patient, and one patient will improve on thyroid, another on thymus, and another, perhaps, on epinephrin. In these cases I have tried first thyroid and then thymus, and finally a combination of pituitary extract and epinephrin. The reason is that if you begin any glandular therapy at all, thyroid seems to be the activator of all, and you may get results from minute doses of this gland. One tenth grain for a week or two will sometimes serve as an activator of the entire system. Then wait a month and give another course of treatment. In this case it was a combination, and on this treatment the young man's conditon is improving since November, when treatment began. It is too short a time, however, to state definitely in regard to results, for the improvement may be due to a remission, and up to the present time the condition has been rather benign in character. Regarding adiposity in the case of the girl, it is impossible to say whether the cerebral condition was secondary to the acute condition producing the dystrophy, or whether probably due to secondary hydrocephalus. This girl showed no symptoms of secondary hydrocephalus. In fact, neurologically she was practically negative —no indigestion, no nausea, no dizziness, no cranial nerve symptoms whatever, simply the symptoms attendant on muscular dystrophy. The roentgenograms in the case show a distinct atrophic bony condition of the cranial vault, probably eroded in parts; the bony structure itself is thin and friable. That is probably not due to any particular internal hydrocephalus, but is actually part of the syndrome itself.

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